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TOTAL UTERINE SAMPLING*

A Proposed Aid in the Early Cytologic Detection of Cancer

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MOST gynecologists agree that early cervical cancer is rarely visualized in clinical practice. The early phase in the history of the disease is preclinical. The vaginal smear has presented abundant evidence of this concept. Papanicolaou,[1, 2] Meigs,[3] Jones[4] and others have reported cancer cells in vaginal secretions in women whose cervixes appeared clinically normal. Traut[5] has insisted, with good reason, that the vaginal smear represents the most valuable clinical test in the armamentarium of the gynecologist. Few gynecologists order vaginal smears on patients whose cervixes look normal, and yet the most important positive findings come from this group. Ayre[6] has pointed out that Schiller's statement, "100% of epidermoid carcinoma of the cervix are from the squamous margin of the

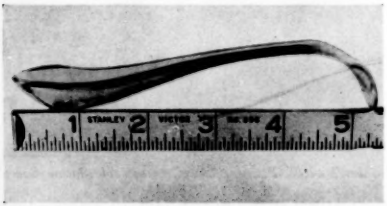

FIGURE 1. *Photograph of the Cervical Spoon.*

squamo-columnar junction," means that cancer arises from a known spot no larger than a dime with greater frequency than any similar area in the human anatomy. That squamous-cell cancer has this point of origin, Novak,[7] TeLinde[8] and Taylor[9] all agree. Scraping of the squamocolumnar junction may not injure the malignant zone sufficiently to induce obvious bleeding from this site, in marked contrast to the well known behavior when a friable, clinical cancer is traumatized. Ayre[10] has refined the method of acquiring the cytologic material by surface scraping by a spatula, showing that even in the nonexfoliative stage

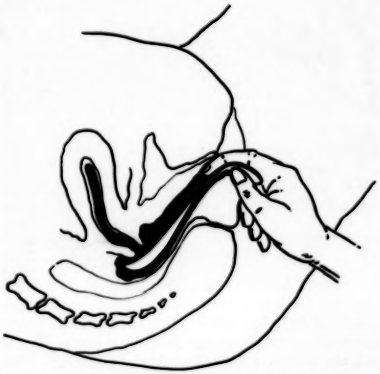

FIGURE 2. *Diagram, Showing the Spoon in the Cervical Canal. The shaft of the spoon is pressed beneath the cervix.*

adequate concentration of cancer cells may be so attained.

Younge, Hertig and Armstrong[11] report that carcinoma in situ involving the surface only and not the cervical lymph nodes can be revealed by vaginal smear in only 53 per cent of cases. Moreover, endometrial and endocervical adenocarcinomas do not exfoliate into the vaginal secretion readily. Fremont-Smith and Graham[12] state that in 6 of 39 cases of cancer of the endometrium no cancer cells could be found in the smears. The high percentage of positive cases missed was attributed chiefly to the fact that

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only a single set of smears was studied. It is obvious that if a method were devised to collect exfoliative samples over a certain period not only cervical but also endometrial and endocervical carcinoma could be diagnosed by the same method.

In menstruating women, nature presents a spontaneous uterine sample consisting of endometrium of the stratum compactum. Gynecologists interested in ovarian function and fertility studies have utilized this type of material by doing menstrual biopsies with the curette on the first day of the cycle. However, because of the practical difficulty of determining the hour of onset of menstrual flow, it is often impossible to collect truly initial menstrual histologic material by office biopsy. It has been known for some time that at the onset of menstruation, the cervical os is soft and patulous and gapes readily. At this time, therefore, nature presents a modification of the normal anatomy and physiology, and sampling of the endocervical cytology, squamocolumnar cytology and endometrial cytology can be most readily achieved. A simple method of total uterine sampling at this time is presented below. Its usefulness as a screening method for the early detection of malignant changes is an obvious corollary. Exfoliative endometrium and scraped endocervical and stratocolumnar-junction tissue in quantitatively substantial samplings are easily obtained in the office.

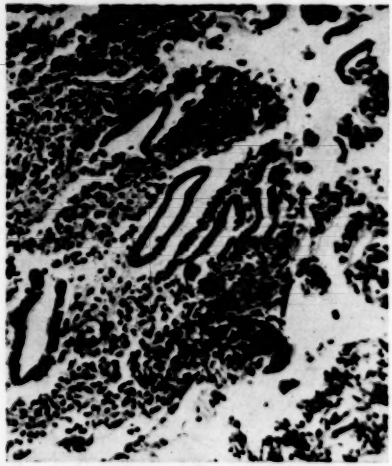

FIGURE 3. *Low-Power Photomicrograph of Spoon Sample of Endometrium (x250).*

Note the compact layer, with good predecidual stroma, in contrast to the necrotic spongiosa layer.

The cervical spoon[12] (Fig. 1) is a lucite plastic device designed to fit the posterior vaginal fornix beneath the cervix. It has a short, curved handle, which fits down against the perineum, where it can be anchored with small strips of cellophane tape. With the labia spread by means of one hand, the spoon is inserted by pressure on its shaft beneath the cervix with the other index finger (Fig. 2). The sensation has been described as analogous to that produced by wearing a Smith pessary. It is neither painful nor uncomfortable. The spoon may be worn throughout the day after Tampax has been placed within the introitus to absorb the menstrual fluid. In the 4-cc. cup of the spoon the endometrial exfoliative fragments, which are larger than those usually obtained by office curettage, will collect without necrosis from the vaginal acidity. The Tampax may be changed hourly, and the endometrial fragments swirled off in physiologic saline solution. After sedimentation by standing for ten minutes the supernatant saline solution with suspended red cells is decanted. The residual endometrial fragments are then deposited in a small jar of 10 per cent formalin or other fixative solution for the usual cell-block studies. When blood loss is minimal the fragments can be placed directly in

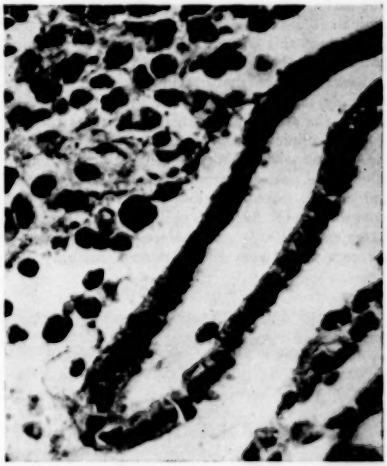

FIGURE 4. *High-Power Photomicrograph of Spoon Sample of Endometrium (x500).*

Note the good predecidual stroma and secretory exhaustion of tortuous glandular mucosa.

formalin. Ample cytologic samples may be obtained during the first twelve to twenty hours of flow. As an overnight technic when the onset of menstruation occurs in the late evening this method has the obvious advantage that the patient may bring to the office initial endometrial material, which is often lost in the interim (Fig. 3 and 4).

When the patient is seen in the office, speculum examination often reveals endometrial fragments entangled in the mucocervical discharge. The gaping cervical os discloses endocervical areas, which may then be readily gently scraped with the rim of the spoon sharpened by a nail file. The squamocervical and endometrial curettage, is an essential supplementary procedure before therapy is considered. I prefer my own combination spoon and curet. The small spoon end cuts a surface-stratocolumnar biopsy. The curet end obtains endocervical and endometrial samples with minimal discomfort.

Figure 6 shows a cervical smear demonstrating mottled cytoplasm and hyperchromatic nuclei typical of squamous-cell carcinoma.

A screening method of detecting patients who need further study for carcinoma must be simple, inexpensive and acceptable to the fastidious middle-aged woman. All are familiar with the apathy and discouragement of the patient who has had repeated smears, biopsies and curettages. In addition

FIGURE 5. *High-Power Photomicrograph of Spoon Sample of Cervix (x500).*

There is marked metaplasia and hypertrophy of the cervical epithelium, indicating necessity for biopsy.

columnar junction may be similarly scraped circumferentially (Fig. 5). The scrapings are then swirled off from the spoon directly into the formalin for cell-block study, or smeared on glass slides and placed in ether-alcohol solution for study by the Papanicolaou method.

However, a positive cervical or vaginal smear does not constitute a diagnosis of cancer. Surface biopsy, either circular, by cone knife,[14] spoon[15] or clamp,[16] or by multiple excision, as well as endo-

FIGURE 6. *Cervical Smear, Showing Mottled Cytoplasm and Hyperchromatic Nuclei Typical of Squamous-Cell Carcinoma.*

the expense involved may be a source of embarrassment to both the patient and the physician.

In the post-menopausal patient with dubious smears and biopsies continuous overnight collection is possible. The spoon's contents are immersed directly into the formalin solution for cell-block study. Only 10 per cent of cancer of the cervix is Stage I when detected. By routine screening of women over thirty-five every six months this therapeutically hopeful group can be enlarged.*

SUMMARY

Total cytologic sampling from the cervix, endocervix and endometrium by the cervical spoon on the first day of the menstrual cycle is described.

*The method described above is now being employed in a cancer-screening survey at Cambridge City and St. Elizabeth's hospitals.

The method is simple and inexpensive.

Post-menopausal, continuous cervicouterine exfoliation can be collected.

An early screening aid to the diagnosis of cancer is available as a simple office procedure.

Biopsy must supplement smears before the final diagnosis is made and therapy started.

I am indebted to Dr. O. J. Pollak, of the Quincy City Hospital, for the preparation of the photomicrographs.

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References

1. Papanicolaou, G. N. New cancer diagnosis. In *Proceedings of the Third Race Betterment Conference, January 2-6, 1928.* 748 pp. Battle Creek, Mich.: Race Betterment Foundation 1928. P. 538.
2. Papanicolaou, G. N., and Traut, H. F. *Diagnosis of Uterine Cancer by the Vaginal Smear.* 47 pp. New York: Commonwealth Fund, 1943.
3. Meigs, J. V., Graham, R. M., Fremont-Smith, M., Kapnick, I., and Rawson, R. Value of vaginal smear in diagnosis of uterine cancer. *Surg., Gynec. & Obst.* 77:449-461, 1943.
4. Jones, C. A., Neustaedter, T., and MacKenzie, L. L. Value of vaginal smears in diagnosis of early malignancy. *Am. J. Obst. & Gynec.* 49:159-168, 1945.
5. Traut, H. F. Value of stained vaginal smear in gynecological diagnosis. *Rocky Mountain M. J.* 44:376-380, 1947.
6. Ayre, J. E. Vaginal smear: precancer cell studies using modified technique. *Am. J. Obst. & Gynec.* 58:1205-1219, 1949.
7. Novak, E. Pseudomalignant and precancerous lesions of cervix. *J. A. M. A.* 100:1145-1151, 1937.
8. TeLinde, R. W., and Galvin, G. A. Minimal histological changes in biopsies to justify diagnosis of cervical cancer. *Am. J. Obst. & Gynec.* 48:774-797, 1944.
9. Taylor, H. C., Jr., and Guyer, H. B. Seven-year history in early cervical cancer. *Am. J. Obst. & Gynec.* 52:451-455, 1946.
10. Ayre, J. E. Diagnosis of preclinical cancer of cervix: cervical cone knife: its use in patients with positive vaginal smear. *J. A. M. A.* 138:11-13, 1948.
11. Younge, P. A., Hertig, A. T., and Armstrong, D. Study of 135 cases of carcinoma in situ of cervix at Free Hospital for Women. *Am. J. Obst. & Gynec.* 58:867-895, 1949.
12. Fremont-Smith, M., and Graham, R. M. Diagnosis of uterine cancer by vaginal smear. In *Progress in Gynecology.* Edited by J. V. Meigs and S. H. Sturgis. 552 pp. New York: Grune & Stratton, 1946. Pp. 92-94.
13. Doyle, J. B. Cervical spoon: aid to spermigration and semen sampling: preliminary report. *Bull. New Eng. M. Center* 10:225-231, 1948.
14. Ayre, J. E. Cervical cytology in diagnosis of early cancer. *J. A. M. A.* 136:513-517, 1948.
15. Novak, E. What constitutes adequate cancer detection examination of cervix. *Am. J. Obst. & Gynec.* 58:851-866, 1949.
16. Gusberg, S. B. Detection of early carcinoma of cervix; coning biopsy. *Am. J. Obst. & Gynec.* 57:752-756, 1949.

AN EVALUATION OF REPORTED POISONINGS BY ACETYLSALICYLIC ACID*

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THE salicylates have been used medicinally since ancient times. Hippocrates described them, and their effects are reported in the earliest works of the modern medical period. In recent years the production of just one of the salicylates — acetylsalicylic acid — in the United States has been greater than that of any other medicinal drug intended for internal use — exceeding 9,000,000 pounds in one year. This drug is today among the most widely used of all medicaments, particularly as an analgesic but also as an antipyretic and in the treatment of rheumatic fever. The large amount of acetylsalicylic acid consumed and the variety of its therapeutic uses call for a sound evaluation of its safety based on the scientific literature that it has evoked.

Acetylsalicylic acid, like most drugs that are useful in therapeutic doses, causes toxic symptoms if sufficiently large amounts are taken; the severity of the symptoms increases as the dosage is further increased. The symptoms vary from mild nausea, vertigo or tinnitus to serious collapse, unconsciousness, coma or death. In a review of hospital records[1] the following symptoms of acetylsalicylic acid toxicosis were observed: dizziness, tinnitus, deafness, nausea, vomiting, fever, prostration, rapid pulse, profuse perspiration in severe cases sometimes accompanied by subnormal temperature, restlessness and coma. In severe cases there was also a decrease in the alkali reserve of the blood, presence of albumin, casts and red blood cells in the urine, and frequently acetone in the urine.

Besides the general toxic effects of acetylsalicylic acid, idiosyncrasy to the drug has been observed in some allergic persons. In these cases the symptoms are evoked by small amounts and consist of typical allergic manifestations such as skin eruptions, edema, asthma, vasomotor disturbances, headache, perspiration, salivation, palpitation, syncope and collapse. Idiosyncrasy, however, is relatively infrequent. From a thorough study of the subject, Gardner and Blanton[2] concluded that the incidence in the general population in approximately 2 per 1000, and that of these only a small proportion have severe reactions.

The effects of acetylsalicylic acid on individual organs and functions of the body have been investigated extensively. In the study of these effects on the cardiovascular system, perfusion of the isolated heart has often been employed. Only extremely high concentrations of the neutralized acid, far in excess of those found in vivo, were observed to depress cardiac action.[3-5] The erroneous concept that acetylsalicylate has an intensely toxic action on the isolated heart, even in concentrations occurring in the blood of persons taking small therapeutic doses, arose from perfusion of the heart with modified Ringer's solution from which sodium bicarbonate was omitted, thus preventing the neutralization of the acetylsalicylic acid.[6] In the absence of a neutralizing base in the perfusing fluid the toxic effects observed were due to the acidity

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and not to the salicylate. Only a neutral salt of acetylsalicylic acid can exist in the blood in vivo.

The influence of acetylsalicylic acid on cardiovascular function in the intact animal has a closer bearing on practical toxicology. No effects, or only a mild depression of the pulse rate or blood pressure, have been observed in animals, even with toxic amounts.[4, 7-9] Therapeutic doses have no effect on cardiac action in man.[10]

No significant effects of small amounts of acetylsalicylic acid on respiration or on the acid-base equilibrium of the blood have ever been reported. Marked consequences of large amounts have often been observed in animal experiments and in man.[9, 11-13] These have consisted of alterations in the volume of breathing, changes in the carbon dioxide content and the alkali reserve of the blood, changes in the pH of the blood and in the acidity of the urine and alterations in the urinary excretion of ammonia and urea. The earlier interpretation of these effects was that the salicylate causes an acidosis; more recent observations,[13, 14] however, have shown that the drug, in large amounts, is a respiratory stimulant and produces a respiratory alkalosis.

Functional disturbances of the gastrointestinal tract such as nausea, gastric discomfort and vomiting have frequently been reported, particularly after large doses.[7, 15-21] These disturbances have been reported as less severe than those caused by other salicylates[15-17] and have been found not to persist even on continued use of the drug,[22] and their occurrence after parenteral administration of large amounts of salicylate indicates that they may be due to a central action as well as to local irritation.

A number of reports have described gastric ulcers and bleeding in animals after the administration of large and toxic doses of acetylsalicylic acid orally[9, 22-26] and parenterally.[27] A few cases of gastric irritation and bleeding in man following the use of small doses have also been reported.[28, 29] Contrary to the latter observations, however, Paul[21] found no hyperemia, edema or hemorrhage in 107 subjects, each given 1.0 gm. (15 gr.) of the drug.

It may be concluded from the literature that large amounts of acetylsalicylic acid frequently cause gastrointestinal disturbances; small therapeutic doses rarely do so, and only in sensitive persons.

There is no indication that small therapeutic doses have any effect on the kidneys or on renal function. Large therapeutic doses of salicylate, such as those employed in the treatment of rheumatic fever, may cause renal impairment and albumin or casts in the urine,[30-35] which disappear without residual damage even though salicylate therapy is continued.[21, 32] Massive doses given to animals have resulted in nephritis.[19, 22, 24, 36-39]

Tinnitus and slight deafness are common symptoms of salicylism. In the treatment of rheumatic fever, prior to the routine clinical use of blood analysis to determine the concentration of salicylate present, the optimum rate of administration of salicylates was often judged by the appearance of these symptoms. There is only meager evidence, however, that small doses, as used for analgesia, affect the auditory function,[40-42] and there is no evidence that such amounts are injurious to the ear even when taken repeatedly.

A possible relation between intensive salicylate medication and bleeding has been repeatedly suggested in the literature.[43-45] In the past few years many investigators have reported a prothrombinopenic effect of salicylate in man.[46-60] In most of these reports the doses were high, and the administrations of large therapeutic doses of acetylsalicylic acid and repeated administration of small amounts were not found to have a prothrombinopenic effect.[48, 53, 61] There is little doubt, from present knowledge, that salicylate in sufficiently large dosage has a prothrombinopenic action in man, and that hypoprothrombinemia is associated with a prolonged bleeding and clotting time. This effect may be a contributing factor in the hemorrhages observed in diseases for which salicylate has been given in large and repeated doses. The prothrombinopenic effect, however, is limited to large doses, repeatedly administered over a prolonged period, and there is no evidence that small doses as used for analgesia cause any clinically significant hypoprothrombinemia.

These data concerning the effects of large amounts of acetylsalicylic acid on specific organs and functions offer a rational explanation for many of the observed symptoms of salicylate intoxication, but they provide little information concerning the practical question of safety or danger in the therapeutic use of the drug. As a source of such information, the experience of a vast number of people who have consumed enormous quantities of the drug annually for many years is unequaled. A statistical and case study of the reported instances of acetylsalicylic acid poisoning can therefore provide the best information concerning any dangers inherent in the therapeutic use of this drug.

Reports of such poisoning, however, are not abundant. Undoubtedly, the appearance of mild toxic symptoms has frequently been observed in the treatment of rheumatic fever, in which the largest dosages are used. During such therapy these symptoms have often been accepted as indicative only of adequate dosage and hence have been ignored. There are few reported cases of death associated with the treatment of rheumatic fever.

Only 88 cases of poisoning by acetylsalicylic acid have been reported from which such data as the dosage, duration of administration, severity of symptoms and reason for taking the drug may be obtained. A summary and analysis of these data

have been made[62] and are shown in Tables 1, 2, 3 and 4.

One striking fact apparent from these summarizations is that in a third of the reported cases of poisoning acetylsalicylic acid was taken in attempted suicide. Since massive amounts of drug are usually consumed in such attempts, it is not surprising that in more than 58 per cent of all the cases extremely large amounts of acetylsalicylic acid were consumed (Table 2). The large majority of poisonings occurred after a few repeated doses during a short period (Table 3). Fifty-four per cent of the poisonings were graded as severe or terminated fatally. These two findings are compatible with the fact that in the majority of cases large doses of the drug were consumed. The small number of poisonings reported from prolonged, repeated consumption (Table 3) indicates the relative safety of such use of therapeutic amounts of the drug.

Certain conclusions seem obvious. A majority of the reported poisonings by acetylsalicylic acid were due to the consumption of excessive amounts of the drug, most often in attempted suicide. The symptoms in these cases were severe, or there was a fatal outcome. In the remaining cases, involving small or moderate amounts, the symptoms were correspondingly mild. In striking contrast to the disregard of mild toxic symptoms from acetylsalicylic acid in rheumatic-fever therapy is the indiscriminate application, in these cases, of the term "poisoning" to effects ranging from mild vertigo or nausea to serious collapse, coma and death.

The lethal dose of a drug is an important consideration in the evaluation of its safety for therapeutic use. The lethal dose of acetylsalicylic acid for man is largely a matter of opinion based on unavoidably unreliable evidence. Few cases of fatal poisoning have occurred in which the administration was reliably observed by a physician and the exact dose known. In most cases the dose was approximated by circumstantial evidence such as an empty bottle thought to have contained some amount of the drug or, more often, the word of the family that the victim took a certain number of tablets during the day. Such evidence has little quantitative validity.

Another point of uncertainty concerning the lethal dose of acetylsalicylic acid lies in the designation of the cause of death as due to this drug. In many cases, as when a person is acutely ill and dies after a seemingly small dose of the drug, the decision whether the acetylsalicylic acid was the main or a contributing cause or of no effect is wholly a matter of opinion. This uncertainty is reflected in the textbooks on toxicology. Thus, McNally[63] states that the lethal doses reported for man show wide variation. He states also that acetylsalicylic acid is about half as toxic as sodium salicylate and that he has never seen a fatal case from an overdose of acetylsalicylic acid. Goodman and Gilman[64] and Sollmann[65] likewise give no values for the lethal dose but merely state that the drug is more toxic than sodium salicylate.

The only other sources of information concerning the possible danger of fatal poisoning in the use of acetylsalicylic acid are the published reports of deaths attributed to the drug. These reports consist of three general types: records of the United States Bureau of the Census; statistical summaries of fatal cases reported in the literature; and reports of individual cases of death following the use of acetylsalicylic acid.

TABLE 1. *Reasons for Taking Acetylsalicylic Acid.*

REASON	NO. OF CASES	PERCENTAGE
Rheumatic fever	16	22.2
Analgesia	17	23.6
Other medicinal use	10	13.9
Accidents	5	7.0
Suicidal attempts	24	33.3
Not stated	16	—

TABLE 2. *Doses of Acetylsalicylic Acid Taken.*

DOSE gm.	NO. OF CASES	PERCENTAGE
Up to 1.5	14	19.7
1.5 to 12.0	19	26.8
Over 12	38	53.5
Not stated	17	—

TABLE 3. *Period over Which Acetylsalicylic Acid Was Taken.*

PERIOD	NO. OF CASES	PERCENTAGE
(Single dose)	16	25.8
1 day	22	35.5
Up to 1 wk.	12	19.4
Over 1 wk. and up to 1 mo.	3	4.8
Over 1 mo. and up to 1 yr.	2	3.2
More than 1 yr.	4	6.5
Duration unknown	3	4.8

TABLE 4. *Severity of Intoxication.*

INTOXICATION	NO. OF CASES	PERCENTAGE
Slight	18	20.5
Moderate	22	25.0
Severe	17	19.3
Fatal	31	35.2

The records of the United States Bureau of the Census provide statistics on deaths reported as due to acetylsalicylic acid in the years 1926, 1928 and 1930 through 1943. Since 1933 the death-registration system of the Bureau has included the 48 states. The data from these records are summarized in Table 5. The number of deaths and the rate per 1,000,000 population are low and show no trend over the entire period. During the period 1939 through 1943 in the United States the average annual total mortality from accidental poisoning by all solids and liquids was reported as 1267, of which about 1 per cent was attributed to acetylsalicylic acid. This low incidence is striking in view of the extensive use and wide availability of the drug.

Besides being relatively few in number, the deaths attributed to acetylsalicylic acid in the statistics of the Census Bureau undoubtedly include many in which a fatal disease or other cause of death was present, so as to render the toxic symptoms of the drug only incidental. In such cases, even if the certifying physician reports an acute or chronic disease or some other factor as the primary cause of death, acetylsalicylic acid poisoning will nevertheless always be recorded as the primary cause in the official statistics. This procedure is in accord with the rule governing the statistical handling of death certifications, under which acute poisoning by solids and liquids supersedes nearly all other causes. Thus, over-reporting of deaths due to poisonings is likely in the Census reports.

Statistical summarizations of deaths attributed to acetylsalicylic acid have been reported by a few investigators. In 1934 Lowy[66] submitted questionnaires to hospitals in the United States concerning deaths attributed to antipyretics. In 2046 hospitals with a total of annual admissions approximating 2,500,000, 4 deaths were attributed to acetylsalicylic acid. This rate is low and corresponds to that reported in the data of the Census Bureau. No data were given concerning the dosages of acetylsalicylic acid involved in these deaths.

In a review of the records of 752 patients hospitalized in Hungary who had taken large amounts of acetylsalicylic acid, Balázs[1] found only 4 deaths. This report, of course, yields no information concerning the per capita incidence in the population of deaths due to the drug. The report showed, however, that only huge doses of 30 to 40 gm. were fatal.

In 1940 the *Lancet*[67] reported the occurrence in England and Wales of 33 deaths attributed to acetylsalicylic acid in 1936, 61 in 1937 and 65 in 1938. From the same areas Hopkins[68] reported the fatal cases attributed to acetylsalicylic acid for the years 1938 to 1942 as recorded by the Registrar General. There were 272 deaths during this period. Although this number is high, suicide with massive amounts of the drug accounted for 75 per cent of the incidence.

In contrast to the data of the Bureau of the Census and the summary reports, much information may be obtained, from individual reports of fatal cases, concerning the amount of acetylsalicylic acid consumed, the reason for taking the drug and the state of health of the person at the time the drug was taken. Thirty-one cases of death attributed to acetylsalicylic acid have been reported[69-89] with clinical data offering such information.

Of these 31 cases, 11 were cases of suicide in which quantities of the drug ranging from 20 to 75 gm. were taken at one time. In 1 case of suicide, in which the amount of acetylsalicylic acid taken was unknown, the victim was an aged woman who, at autopsy, was found to have advanced arteriosclerosis and aortic insufficiency. Another person jumped into a pool immediately after taking the drug and was drowned.

In 6 fatal cases, not reported as suicides, large to massive amounts of acetylsalicylic acid were known to have been taken. Two of these persons had histories of mental disease. Another took 50 gm. of the drug, supposedly for a toothache. Still another, acutely ill with influenza, found access to and consumed 60 gm. (1000 gr.) of acetylsalicylic acid.

In 9 cases of death attributed to acetylsalicylic acid the amounts of drug taken were not known. In 2 of these the contents of the viscera indicated that large amounts of salicylate had been consumed. Two of these persons had histories of mental illness. In 2 cases a serious underlying disease was present, in 1 there was an unspecified ailment and in 1 other drugs were taken simultaneously.

Besides these 31 nonallergic deaths attributed to acetylsalicylic acid, 7 deaths have been reported[90-96] as being due to idiosyncrasy to the drug. In these cases typical allergic manifestations were observed, and death resulted from small doses of the drug. All the victims were asthmatic and had

TABLE 5. *Accidental Deaths from Acetylsalicylic Acid.**

YEAR	TOTAL POPULATION IN MILLIONS	NUMBER	RATE
1926	103.8	17	0.164
1928	113.6	14	0.123
1930	117.2	9	0.077
1931	118.1	12	0.102
1932	118.9	11	0.093
1933	125.6	19	0.151
1934	126.4	13	0.103
1935	127.3	8	0.063
1936	128.1	21	0.164
1937	128.8	13	0.101
1938	129.8	17	0.131
1939	130.9	16	0.122
1940	131.7	18	0.137
1941	133.1	8	0.060
1942	135.8	11	0.082
1943	136.0	20	0.147
Total		227	
Average			0.113

*Data taken from United States Bureau of the Census.

shown hypersensitivity to acetylsalicylic acid on one or more previous occasions.

This review of the reported cases of death attributed to acetylsalicylic acid reveals several noteworthy features aside from the small number of cases. A large portion of them were suicides. These, as well as another large portion that were not suicides, consumed amounts of acetylsalicylic acid far in excess of the doses used therapeutically. In the other cases the amounts were unknown but, from the symptoms observed, can be presumed to have been large. In several cases of poisoning there was a history of mental disorder; in many others there were severe underlying diseases. Severe poisoning, or death from large or massive doses of any drug, whatever the reason for its consumption, is not a criterion of the possible danger in its therapeutic use. Finally, there is a conspicuous absence of reports of serious poisoning or death, other than allergic poisonings, associated with ordinary therapeutic use of acetylsalicylic acid, even when this use has been continued over a long period.

References

1. Balázs, J. Über Acetylsalizylsäure (Aspirin)-Vergiftungen. *Med. Klin.* **36**:1664-1666, 1930.
2. Gardner, E., and Blanton, W. B. Incidence of aspirin hypersensitivity. *Am. J. M. Sc.* **200**:390-394, 1940.
3. Salant, W., and Johnson, R. L. Action of salicylates on isolated heart. *Proc. Soc. Exper. Biol. & Med.* **20**:390, 1923.
4. Mendenhall, W. L., and Camp, H. F. Effect of acetylsalicylic acid on cardiac irritability. *Boston M. & S. J.* **190**:312-314, 1924.
5. Johnston, R. L. Effect of certain salicyl derivatives on isolated hearts of frogs and turtles. *J. Pharmacol. & Exper. Therap.* **57**:193-197. 1936.
6. Simon, I. Ricerche farmacologiche sull'acido acetilsalicilico. *Arch. di farmacol. sper.* **55**:151-182, 1933.
7. Chidichimo, F. Azione fisiologica e terapeutica dell'aspirina con speciale riguardo all'utero. *Ann. di ostet.* **27**:356-374, 1905.
8. Block, F. *Untersuchungen Über Aspirin, Novaspirin und Aspirophen. Leipzig thesis*, 1909.
9. Dodd, K., Minot, A. S., and Arena, J. M. Salicylate poisoning: explanation of more serious manifestations. *Am. J. Dis. Child.* **53**: 1435-1446, 1937.
10. Barbour, H. G., and Devenis, M. M. Antipyretics. II. Acetylsalicylic acid and heart regulation in normal individuals. *Arch. Int. Med.* **24**:617-623, 1919.
11. Johnson, C. C. Salicylates. XIX. Question of acidosis following administration of salicylates. *J. A. M. A.* **94**:784-789, 1930.
12. Odin, M. Is salicyl-poisoning "acidosis"? *Acta med. Scandinav. Supp.* **50**:177-186, 1932.
13. Andersen, A. H., Andersen, E., and Brun, G. C. Mechanism of toxic action of salicylic acid. *Acta med. Scandinav.* **109**:336-354, 1941.
14. Ryder, H. W., Shaver, M., and Ferris, E. B., Jr. Salicylism accompanied by respiratory alkalosis and toxic encephalopathy: report of fatal case. *New Eng. J. Med.* **232**:617-621, 1945.
15. Gazert. Therapeutische Erfahrungen mit Aspirin. *Deutsch. Arch. f. klin. Med.* **68**:142-154, 1900.
16. Görzes. Über neuere Arzneimittel: Aspirin und Digitalis-Dialysat. *Berl. klin. Wchnschr.* **39**:753-755, 1902.
17. Roch, M. Acide acétyl-salicylique et salicylate de soude. *Bull. gén. de thérap.* **163**:218-223, 1912.
18. Barbour, H. G., and Lozinsky, E. Nontoxicity and antipyretic efficiency of tolysin (ethyl ester of paramethylphenylcinchoninic acid). *J. Lab. & Clin. Med.* **8**:217-226, 1923.
19. Thompson, H. E., and Dragstedt, C. A. Chemical and pharmacological properties of calcium acetylsalicylate. *J. Am. Pharm. A.* **22**:1096-1101, 1933.
20. *Idem.* Modifying action of calcium and sodium bicarbonate on salicylate intoxication. *Arch. Int. Med.* **54**:308-312, 1934.
21. Paul, W. D. Effect of acetylsalicylic acid (aspirin) on gastric mucosa: gastroscopic study. *J. Iowa M. Soc.* **33**:155-158, 1943.
22. Keith, J. D., and Ross, A. Observations on salicylate therapy in rheumatic fever. *Canad. M. A. J.* **52**:554-559, 1945.
23. Chistoni, A., and Lapresa, F. Ricerche farmacologiche sull'aspirina. *Arch. di farmacol. sper.* **8**:63-80, 1909.
24. Busacchi, L. Lesioni istologiche da acido acetilsalicilico e da acetilsalicilato di sodio: richerche sperimentali. *Rass. di Ziopat. clin. e terap.* **10**:301-311, 1938.
25. Shimamura, M., and Aoki, A. Über das durch Aspirin hervorgerufene Magengeschwür. *Folia pharmacol. japon.* **27**:19, 1939.
26. Stutzman, J. W., Orth, O. S., and Mellish, C. H. Comparison of calcium acetylsalicylate (as calsamate) to acetylsalicylic acid. *J. Pharmacol. & Exper. Therap.* **73**:420-430, 1941.
27. Barbour, H. G., and Dickerson, V. C. Gastric ulceration produced in rats by oral and subcutaneous aspirin. *Arch. internat. pharmacodyn. et de thérap.* **58**:78-87, 1938.
28. Douthwaite, A. H., and Lintott, G. A. M. Gastroscopic observation of effect of aspirin and certain other substances on stomach. *Lancet* **2**:1222-1225, 1938.
29. Hurst, A., and Lintott, G. A. M. Aspirin as cause of haematemesis: clinical and gastroscopic study. *Guy's Hosp. Rep.* **89**:173-176, 1939.
30. Lüthje, H. Über die Wirkung von Salicylpräparaten auf die Harnwege nebst einigen Bemerkungen über die Genese der Cylinder und Cylindroide. *Deutsch. Arch. f. klin. Med.* **74**:163-206, 1902.
31. Klieneberger, C., and Oxenius, R. Über Urine und Urinsedimente bei normalen Personen, bei rheumatischen Erkrankungen und nach der Einwirkung von Salicylpräparaten. *Deutsch. Arch. f. klin. Med.* **80**:225-244, 1904.
32. Knecht, E. Über die Wirkung des Natrium salicylicum auf den Harnapparat. *München. med. Wchnschr.* **51**:956-960, 1904.
33. Frey, E. Die Vermeidung der Nierenreizung nach grossen Salizylgaben. *München. med. Wchnschr.* **52**:1326-1329, 1905.
34. Ehrmann, R. Über Albuminurie und über die Ausscheidungsverhältnisse der Salicylsäure aus dem Organismus von Gesunden und Gelenkrheumatikern. *München. med. Wchnschr.* **54**:2595-2597, 1907.
35. Scott, R. W., Reycraft, J. L., and Hanzlik, P. J. Salicylate edema. *J. Pharmacol. & Exper. Therap.* **9**:348, 1917.
36. Scott, R. W., and Hanzlik, P. J. Salicylates. III. Salicylate albuminuria. *J. A. M. A.* **67**:1838-1842, 1916.
37. Hanzlik, P. J., and Karsner, H. Salicylates. VI. Renal, functional and morphologic changes in animals following administration of salicylate. *Arch. Int. Med.* **19**:1016-1028, 1917. VII. Further observations on albuminuria and renal functional changes following administration of full therapeutic doses of salicylate. *Ibid.* **19**: 1029-1041, 1917.
38. Barbour, H. G., and Porter, J. A. Investigation of possible protective action of calcium gluconate in aspirin poisoning. *J. Pharmacol. & Exper. Therap.* **60**:224-227, 1937.
39. Taylor, H. M. Deafness from drugs and chemical poisons. In *Medicine of the Ear.* Edited by E. P. Fowler, Jr. 590 pp. New York: Thomas Nelson & Sons, 1939.
40. Macht, D. I., Greenberg, J., and Isaacs, S. Effect of some antipyretics on acuity of hearing. *J. Pharmacol. & Exper. Therap.* **15**: 149-165, 1920.
41. Wikler, A., Goodell, H., and Wolff, H. G. Studies on pain: effects of analgesic agents on sensations other than pain. *J. Pharmacol. & Exper. Therap.* **83**:294-299, 1945.
42. Falbe-Hansen, J. Clinical and experimental histological studies on effects of salicylate and quinine on ear. *Acta oto-laryng. Supp.* **44**:1-216, 1941.
43. Balette, E. *De l'action du salicylate de soude sur l'uterus.* 62 pp. Paris: A. Parent, 1883.
44. Fischer, E. Effets secondaires de l'aspirine. *Arch. internat. de laryngol.* **2**:136, 1905.
45. Link, K. P. Anticoagulant from spoiled sweet clover hay. *Harvey Lect.* **39**:162-216, 1944.
46. Butt, H. R., et al. Studies in rheumatic fever. I. Physiologic effect of sodium salicylate on human being, with particular reference to prothrombin level of blood and effect of hepatic parenchyma. *J. A. M. A.* **128**:1195-1200, 1945.
47. Clausen, F. W., and Jager, B. V. Relation of plasma salicylate level to degree of hypoprothrombinemia. *J. Lab. & Clin. Med.* **31**: 428-436, 1946.
48. Coombs, F. S., Jr., Higley, C. S., and Warren, H. A. Magnitude of salicylate hypoprothrombinemia. *Proc. Central Soc. Clin. Research* **18**:68, 1945.
49. Fashena, G. J., and Walker, J. N. Salicylic intoxication: studies on effects of sodium salicylate on prothrombin time and alkali reserve. *Am. J. Dis. Child.* **68**:360-375, 1944.
50. Lawson, R. B. Salicylate therapy for rheumatic fever and atrophic arthritis (Still's disease) in children. *North Carolina M. J.* **5**: 477-482, 1944.
51. Link, K. P., Overman, R. S., Sullivan, W. R., Huebner, C. F., and Scheel, L. D. Studies on hemorrhagic sweet clover disease. XI. Hypoprothrombinemia in rat induced by salicylic acid. *J. Biol. Chem.* **147**:463-474, 1943.
52. Matheson, J. K. Unusual case of salicylic acid poisoning. *Lancet* **1**: 432, 1906.
53. Meyer, O. O., and Howard, B. Production of hypoprothrombinemia and hypocoagulability of blood with salicylates. *Proc. Soc. Exper. Biol. & Med.* **53**:234-237, 1943.
54. Neivert, H. Late secondary tonsillar hemorrhage. I. Studies of prothrombin and vitamin K. *Arch. Otol.* **42**:14-18, 1945.
55. *Idem.* Salicylate-induced hypoprothrombinemia. *J. A. M. A.* **130**: 733, 1946.
56. Owen, G. C., and Bradford, H. A. Prothrombinopenic effect of massive salicylate therapy in acute rheumatic fever. *Ann. Int. Med.* **25**: 97-102, 1946.
57. Rapoport, S., Wing, M., and Guest, G. M. Hypoprothrombinemia after salicylate administration in man and rabbits. *Proc. Soc. Exper. Biol. & Med.* **53**:40, 1943.
58. Shapiro, S. Studies on prothrombin. VI. Effect of synthetic vitamin K on prothrombinopenia induced by salicylate in man. *J. A. M. A.* **125**:546-548 and 923, 1944.

59. Shapiro, S., Redish, M. H., and Campbell, H. A. Studies on prothrombin: prothrombinopenic effect of salicylates in man. *Proc. Soc. Exper. Biol. & Med.* **53**:251-254, 1943.

60. Warren, H. A., Higley, C. S., and Coombs, F. S. Treatment of acute rheumatic fever. *News Letter, A. A. F. Rheumatic Fever Control Program* **2**:27-29, 1945.

61. di Cló, A. V., and Bay, R. Acción de los compuestos salicilados sobre la protrombinemia humana. *Día méd.* **17**:5-0, 1945.

62. Gross, M., and Greenberg, L. A. *The Salicylates: A critical bibliographic review.* 380 pp. New Haven, Conn.: Hillhouse Press, 1948.

63. McNally, W. D. *Toxicology.* 1022 pp. Chicago: Industrial Medicine, 1937.

64. Goodman, L., and Gilman, A. *The Pharmacological Basis of Therapeutics: A textbook of pharmacology, toxicology and therapeutics for physicians and medical students.* 1383 pp. New York: Macmillan Co., 1941.

65. Sollmann, T. *A Manual of Pharmacology and Its Applications to Therapeutics and Toxicology.* Fifth edition. 1190 pp. Philadelphia: W. B. Saunders Co., 1936.

66. Lowy, O. Comparative study of habitual use of barbiturates and coal-tar derivatives as furnished by reports from various hospital's throughout United States. *Canad. M. A. J.* **31**:638-641, 1934.

67. Annotations. Aspirin as poison. *Lancet* **1**:1091, 1940.

68. Hopkins, P. Aspirin poisoning: report of case. *Lancet* **1**:145, 1945.

69. Lewis, F. W. Case of aspirin poisoning. *Lancet* **1**:64, 1919.

70. Hitch, F. G. Fatal case of poisoning by aspirin. *J. Roy. Nav. M. Serv.* **14**:295-297, 1928.

71. de Jankovich, L. Les empoisonnements morbides par l'aspirine. *Ann. de méd. lég.* **8**:7-10, 1928.

72. Hultkvist, G. Cas mortels d'empoisonnement par l'acide acétylsalicylique. *Acta med. Scandinav.* Supp. **71**:165-169, 1929.

73. Poisoning cases and inquests. *Pharm. J.* **122**:163 and 561, 1929.

74. Balázs, J. Acetylsalizylsäure Vergiftung (Selbstmord). *Samml. Vergiftungsf.* **3**:201, 1932.

75. *Idem.* Acetylsalizylsäure Vergiftung (Selbstmord). *Samml. Vergiftungsf.* **3**:287, 1932.

76. *Idem.* Acetylsalizylsäure Vergiftung (Selbstmord). *Samml. Vergiftungsf.* **5**:25, 1934.

77. Wyllie, A. M. Fatal case of aspirin poisoning, with post-mortem findings. *Lancet* **2**:768, 1935.

78. Eicher, O. Kindesmord durch Aspirin (Gutachten). *Samml. Vergiftungsf.* **7**:25-30 and 65-68, 1936.

79. Neale, A. V. Aspirin poisoning. *Brit. M. J.* **1**:109, 1936.

80. Orzechowski, G. Selbstmord mit Aspirin. *Samml. Vergiftungsf.* **7**: 71, 1936.

81. Biddle, E. Aspirin poisoning. *Brit. M. J.* **1**:1365, 1938.

82. Friedman, J. H. Toxic encephalopathy. Coal tar derivatives as probable etiological factor: report of three cases with neurohistological studies in two. *J. Mt. Sinai Hosp.* **5**:463-471, 1938.

83. Halstrom, F., and Moller, K. O. Ein Fall von tödlicher Salicylsäurevergiftung (Acetylsalicylsäure?) mit quantitativer Salicylsäurebestimmung in den Organen. *Deutsche Zschr. f. d. ges. gerichtl. Med.* **31**:182-188, 1939.

84. Sears, T. P. Excessive use of empirin compound. *Rocky Mountain M. J.* **36**:642, 1939.

85. Medical News. *Brit. M. J.* **1**:135, 1942.

86. Hawkinson, O., and Kerr, E. K. Case of granulocytopenia with severe anemia and staphylococcemia, where only etiological factor was use of aspirin in 40-60 grain doses daily for years. *Illinois M. J.* **83**:168-170, 1943.

87. Ashworth, C. T., and McKemie, J. F. Hemorrhagic complications with death probably from salicylate therapy: report of two cases. *J. A. M. A.* **126**:806-810, 1944.

88. Troll, M. M., and Menten, M. L. Salicylate poisoning: report of four cases. *Am. J. Dis. Child.* **69**:37-43, 1945.

89. Dangerous incompatibility. *Bull. Pharm.* **29**:456, 1915.

90. Vander Veer, A. Asthma problem. *New York M. J.* **112**:392-399, 1920.

91. Wright, C. B. Death from bronchial asthma. *J. A. M. A.* **94**:1218-1221, 1930.

92. Lamson, R. W., and Thomas, R. Some untoward effects of acetylsalicylic acid. *J. A. M. A.* **99**:107, 1932.

93. Macdonald, I. G. Local and constitutional pathology of bronchial asthma. *Ann. Int. Med.* **6**:253-277, 1932.

94. Dysart, B. R. Death following ingestion of five grains of acetylsalicylic acid. *J. A. M. A.* **101**:446, 1933.

95. Francis, N., Ghent, O. T., and Bullen, S. S. Death from ten grains of aspirin. *J. Allergy* **6**:501-505, 1935.

96. Calder, G. Drug allergy. *Univ. West. Ontario M. J.* **9**:107-109, 1939.

ENDOCARDITIS DUE TO *ESCHERICHIA COLI**

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SUBACUTE bacterial endocarditis and various acute forms of endocarditis are usually considered to arise on valves that have been previously damaged. It would be remarkable if endocarditis due to *Escherichia coli* should prove to be an exception, since the colon bacillus is usually an organism of low virulence and one that generally invades only in cases of extensive tissue damage. The explanation for this discrepancy is thought to be due to an increased virulence assumed by *Esch. coli*.[1] A case of this disease is reported below because of the rarity of well documented cases. This case and the seeming incongruity of the colon bacillus attacking a normal valve has led to a review of the position played by rheumatic fever and valve damage in endocarditis due to *Esch. coli*.

CASE REPORT

A 41-year-old Negress entered the emergency room of the hospital on January 17, 1949, in a semicomatose state with a history of severe left-upper-quadrant pain 1 week prior to admission, followed by fever and vomiting of bile-stained material. The pain subsided in about 12 hours, but the patient became drowsy and delirious 2 days before admission. The urine had been scanty.

The patient gave no history of previous febrile illnesses, although she reported having had migratory joint pains in the past 2 years.

The scleras and sublingual mucosa were markedly icteric. The heart was not enlarged, but a systolic murmur was heard over the entire precordium. The abdomen was moderately distended and tense, and there was marked tenderness in the right upper quadrant; the liver edge extended 8 cm. below the right costal margin. On pelvic examination, there was tenderness in both adnexal regions.

The temperature was 101°F., the pulse 32, and the respirations 20. The blood pressure was 96/68.

Urinalyses during the hospital stay showed up to 20 red blood cells per high-power field, with rare white blood cells, in the sediment. There was a trace of albumin on two analyses soon after admission, but no sugar. The red-cell count was 4,800,000 on the day of admission and 2,000,000 3 days prior to death, with hemoglobin values of 13 gm. on admission and 5.5 gm. just before death. The white-cell count, which was 25,000 on the day of admission, fell to 4500 on February 8, but steadily rose after that date to 45,000 3 days prior to death. The Schilling counts showed shifts to the left on all occasions. Agglutinations for typhoid O and H, paratyphoid A, brucella and *Proteus OX-19* were negative; that for paratyphoid B was positive in dilutions of 1:320. Special agglutinations with Type I *Leptospira icterohemorrhagiae* 3 weeks prior to death were reported positive in dilutions of 1:30 and feebly positive in dilutions of 1:100. The nonprotein nitrogen was 240 mg. per 100 cc. on the day of admission but steadily fell to 35 mg. by February 8. The alkaline phosphatase, which was 22 units on admission, fell to 0 units 3 weeks before death and rose to 5.1 units 1 week later. The serum bilirubin was 1.6 mg. per 100 cc. on admission and rose to 1.85 mg. 1 week prior to death; the thymol turbidity on admission was 8 units, rising to 14 units 3 weeks and to 48

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units 1 week before death. A hippuric acid determination 1 week after admission was 1.3 gm., and a bromsulfalein test 2 weeks after admission showed no retention of the dye in 55 minutes. Blood cultures taken on February 9 and February 10 were reported as showing no growth, but cultures on February 11, 12, 17, 18 and 19 all revealed growth of *Esch. coli*. A Kline test was negative.

The clinical impressions of this disease were Weil's disease or an unspecified type of acute hepatitis. The patient was placed on bed rest and given penicillin 100,000 units immediately and 50,000 units every 3 hours during the first 2 weeks. On penicillin therapy, she improved markedly, regained consciousness and began to talk normally; the jaundice became less marked. However, a systolic heart murmur was heard repeatedly throughout the hospital stay. From January 23 to February 8 the temperature remained near normal, and the patient felt well. On February 9 the temperature rose to 104°F., and continued to spike daily. Because of the return of fever, blood cultures were taken, and a needle biopsy of the liver done. The biopsy report was focal necrosis of the liver. On receipt of the blood-culture reports on *Esch. coli*, penicillin was reinstituted and streptomycin, 15 gm. every 6 hours, was started. Despite this therapy, the patient died on February 21, 1949.

At post-mortem examination, performed 2 hours after death, the visceral and parietal pericardia were covered with a finely granular to shaggy exudate, and there was 300 cc. of yellow, cloudy fluid in the pericardial sac. The heart and attached pericardium weighed 545 gm. A large, globose, friable vegetation measuring approximately 1.5 cm. in diameter was located on the posterior semilunar cusp of the aortic valve, extending to involve the aortic surface of the posterior mitral leaflet and the myocardium above the posterior leaf of the tricuspid valve. The chordae tendineae of the mitral valve were moderately shortened and thickened, and there was minimal nodular thickening of the free margin of the anterior leaflet.

The liver weighed 2120 gm., and several dark-brown and yellow areas measuring up to 2 cm. in diameter were noted in the right upper lobe and left inferior lobe. The spleen weighed 240 gm. and showed several soft, white pyramidal infarcts, 1 cm. in diameter. The kidneys weighed 185 and 150 gm. and were swollen, soft and tan. The brain weighed 1320 gm. Several small abscesses 1 mm. in diameter were scattered through the basal ganglions and cortex. An abscess, 1 cm. in diameter, was located in the right globus pallidus and internal capsule.

On microscopical examination sections of the myocardium showed some swelling of the fibers, miliary abscesses and pericapillary infiltration of lymphocytes and a few large mononuclear cells with bar nuclei. The thickened visceral pericardium disclosed interstitial edema and a surface exudate containing many polymorphonuclear cells and lymphocytes. Gram stains of the pericardium revealed no organisms. Sections of the vegetation demonstrated no fibrosis at its base, with peripheral masses of gram-negative bacilli and coccoidal forms.

Sections of liver revealed a pattern of parenchymal necrosis, more marked around the portal areas and the central-vein areas, with some relatively normal mid-zonal portions. Miliary areas of focal accumulation of polymorphonuclear cells were seen, but they revealed no bacteria in gram stains.

Sections of the spleen showed areas of coagulation necrosis containing irregular blue colonies of gram-negative bacilli, surrounded by a zone of polymorphonuclear cells and congested parenchyma. Multiple, small focal collections of mononuclear cells, polymorphonuclear leukocytes and plasma cells were seen in the kidney cortex and collecting-tubule areas, with generalized cloudy swelling of the epithelium of the tubules.

The final pathologic diagnoses were acute bacterial endocarditis due to *Esch. coli*. with vegetation on the aortic valve; serofibrinous pericarditis; acute pyelonephritis; multiple abscesses, with focal necrosis of the liver; infarcts and congestion of the spleen; abscesses of the basal ganglions of the brain; and acute cystitis.

Five ante-mortem blood cultures, and cultures taken at autopsy from the aortic-valve vegetation, and from the liver and gall bladder (combined) showed gram-negative rods. The organisms produced metallic, greenish-bronze colonies on eosin methylene blue plates. On Kliegler's tubes, they gave acid reactions and formed gas on both dextrose and lactose agar, but produced no hydrogen sulfide. These characteristics placed the organism in the *Esch. coli* group. Cultures of the pericardium at autopsy gave no growth.

Discussion

The criteria for acceptance of this case as endocarditis due to *Esch. coli* are the five positive ante-mortem blood cultures, the positive post-mortem cultures of the valve vegetation and the abundant gram-negative rods in the vegetation. Only 12 other cases reported in the literature may be accepted as meeting all or a major part of these qualifications.[1] Five of these 12 were incompletely described by Phipps,[2] inasmuch as positive ante-mortem blood cultures alone were cited. Perusal of the literature since 1946 has failed to reveal any additional case of endocarditis due to *Esch. coli*.

In the case presented above, the history of migratory polyarthritis during the two years prior to the death of the patient suggests rheumatic fever, but the only lesion consistent with a suspicion of rheumatic endocarditis was the minimal thickening of the anterior leaflet of the mitral valve. It is possible that widespread destruction to the valve seen in endocarditis due to *Esch. coli* often obliterates evidence of previous valve damage.

Examination of the case in the University of Arkansas files previously reported in 1946[1] reveals that this patient, a thirty-year-old woman, died after a febrile illness of five months' duration. Her chief complaints were a "streptococcal sore throat and pneumonia." No pathological evidence of rheumatic carditis was found.

Horder's[3] case occurred in a boy aged twelve years who had never suffered from rheumatism or scarlet fever. Of Dickar's[4] 2 patients, 1 was an elderly man with vegetations involving the mitral, aortic and tricuspid valves, and another was a sixty-four-year-old woman with aortic-valve involvement alone, in whose heart several areas of thickening of the free margins of the mitral valve were noted. Dickar gives no statement regarding any positive prior history of rheumatic fever in either of his cases. The valve thickenings in 1 case are suggestive, but, without supporting history or other lesions, especially in a person sixty-four years of age, this cannot be accepted as a positive indication of previous rheumatic fever. Duhig[5] says that his patient was a twenty-five-year-old woman with no history of rheumatic fever, and he gives no pathological evidence of a rheumatic carditis. The case of Crawford and Cruickshank's[6] however, occurred in a forty-three-year-old married woman, who had had scarlet fever in childhood and acute rheumatism at the age of eighteen years. At thirty years of age she began to experience exertional dyspnea, and ankle edema developed about eight months prior to death. Five weeks before death abdominal pain, vomiting, diarrhea, fever and chills developed, persisting until death. At autopsy there was evi-

dence of old endocarditis of the mitral valve, with fibrosis of the cusps and formation of a ridge of thickened tissue with some narrowing of the ring. The chordae tendineae were shortened and thickened. The patient of Harries and Burtenshaw[7] was an eighteen-year-old girl who had an illness simulating chorea two years prior to death. Three weeks before death headache, shivering, vomiting and constipation developed, and she was thought to have typhoid fever, but essentially negative typhoid agglutinations were obtained ten and fourteen days later. The authors reported the finding of acute inflammation of the mitral valve, but no sign of old inflammation or other abnormality. The 5 cases of Phipps[2] were presented without any references of previous history of rheumatic fever or of disease of the valve seen at autopsy.

Only the case of Crawford and Cruickshank[6] may be accepted as giving a history and pathologic evidence of rheumatic carditis. Questionable previous damage to any heart valve was present in one of Dickar's[4] cases and in the case reported above. Excluding from these figures the 5 cases of Phipps, in which no data were given, leaves a positive history of rheumatic fever with pathological confirmation in only 1 of 7 cases, or 14 per cent, and a suggestive history in an additional 2 cases. The indication is that previous rheumatic endocarditis plays a minor role in providing a locus upon which a subsequent *Esch. coli* vegetation may arise.

Felty and Keefer,[8] in their study of 28 cases of bacillemia due to *Esch. coli* and a review of the literature, state that the portals of entry are the urinary tract, the female genital tract and the intestinal tract in that order of frequency. Jacob[9] and Brian[10] rank the portals of entry in cases of septicemia due to *Esch. coli* without endocarditis in descending order of frequency: as the biliary tract, the urinary tract, the gastrointestinal tract and the female genital tract. The initial complaint, in the case reported above, of severe left-upper-quadrant pain of 1 week's duration, supported by the autopsy finding of an acute pyelonephritis, lead one to suspect that the initial portal of entry of the colon bacillus was the urinary tract.

Summary

A proved case of endocarditis due to *Escherichia coli* is presented as the thirteenth such case to be found in the literature and the second reported from this hospital.

It is believed that this disease is the result of increased virulence of the infecting organism, as opposed to other forms of bacterial endocarditis, which usually result from the implantation of the organism on an already damaged valve. The incidence of valve damage and history of rheumatic fever in reported cases are outlined.

The possible points of origin of the organism in bacillemia due to *Esch. coli* are briefly discussed.

References

1. Fletcher, D. E. Bacillus coli endocarditis: review of literature with report of case. *Am. Heart J.* **34**:743-750, 1947.
2. Phipps, C. Acute bacterial endocarditis. *New Eng. J. Med.* **207**: 768-770, 1932.
3. Horder, T. J. Infective endocarditis with analysis of 150 cases and with special reference to chronic form of disease. *Quart. J. Med.* **2**: 289-324, 1909.
4. Dickar, L. Acute bacterial endocarditis due to bacterium acidilactici. *Arch. Int. Med.* **49**:788-798, 1932.
5. Duhig, J. V. Septicaemia and acute infective endocarditis due to Bacterium coli. *M. J. Australia* **1**:435-437, 1933.
6. Crawford, A. M., and Cruickshank, R. Case of infective endocarditis due to bacillus coli. *Glasgow M. J.* **122**:21-24, 1934.
7. Harries, G. E., and Burtenshaw, J. M. L. Acute infective endocarditis due to Bacterium coli. *Lancet* **2**:803, 1937.
8. Felty, A. R., and Keefer, C. S. Bacillus coli sepsis: clinical study of twenty-eight cases of blood stream infection by colon bacillus. *J. A. M. A.* **82**:1430-1433, 1924.
9. Jacob, L. Über Allgemeininfektion durch Bacterium coli commune. *Deutsche Arch. f. klin. Med.* **97**:303-347, 1909.
10. Brian, O. Über Allgemeininfektion durch Bacterium coli commune (Colisepsis). *Deutsche Arch. f. klin. Med.* **106**:379-399, 1912.

SPOROTRICHOSIS IN NEW HAMPSHIRE

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SPOROTRICHOSIS, a disease of mycotic etiology, is caused by *Sporotrichum schenckii*. The disease is common in plants and is more common in animals than in man.[1] In human beings sporotrichosis has been described in a localized lymphangitic form, a disseminated cutaneous form and an extracutaneous form. In its localized lymphatic form, it is characterized by the formation of indolent cutaneous and subcutaneous granulomatous lesions.

FIGURE 1. *Granulomatous Abscesses on the Forearm and Arm.*

The disease was first recognized in 1898 by Schenck, and in the past fifty years has been encountered in increasing frequency. Two hundred and six proved cases of sporotrichosis had been reported in the United States by 1940.[2]

In this country, sporotrichosis is seen most frequently in the Midwest, the West and the Northwest.[3, 4] A review of the literature reveals that the disease is rarely encountered in New England.

To date, 7 cases of sporotrichosis, proved by culture, have been reported in New England. Of these, 4 were reported from Connecticut,[4-6] 2 from Massachusetts,[7, 8] and 1 from Maine.[9] Weise[5] describes the existence of 3 additional unreported, uncultured cases of sporotrichosis in Connecticut.

As far as we can ascertain, this case represents the first case of sporotrichosis reported in the state of New Hampshire.

CASE REPORT

X. X., a 69-year-old-woman, was first seen in the clinic on August 7, 1947. Two and a half months previously a painful, fluctuant swelling of the right index finger developed without any previous trauma of which the patient was aware. The lesion was incised and drained of purulent material. The patient received penicillin, intramuscularly, for 5 days, during which painful, edematous "welts" appeared on the arms and legs. These were of 2 weeks' duration. She complained only of mild chilly sensation. The temperature was reported to have been 100.4°F. by mouth. The finger lesion did not improve, and a second incision and drainage was performed 2 weeks later. One month after the appearance of the initial swelling of the right index finger, a total of eight edematous, erythematous, tender, granulomatous lesions on the right forearm and arm developed. These suppurated within 3 weeks. The original lesion on the right index finger

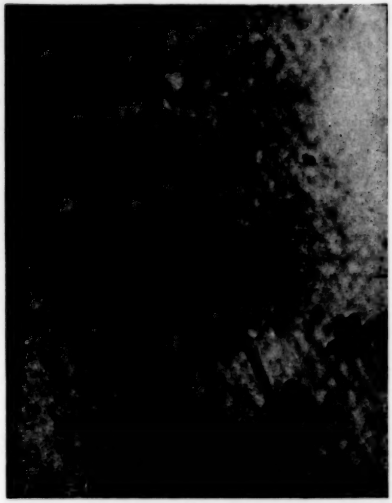

FIGURE 2. *Photomicrograph of* Sporotrichum schenckii *Showing Characteristic Arrangement of the Branching Septate Hyphae-Bearing Conidia in Groups from the Ends of the Conidiophores.*

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completely healed 2 months after its initial appearance. The secondary lesions on the arm and forearm continued to drain purulent material.

The patient had not traveled beyond the limits of central New Hampshire in recent years. She was not aware of having come in contact with any sick animals previous to the onset of her illness. One month before its onset, she had planted rosebushes that she had received by mail from western New York state. Examination and cultural studies of the rosebushes for fungus were not carried out, since the patient had cut them down before this procedure could be instituted.

Physical examination showed eight discrete, infiltrated, fluctuant, pustular, granulomatous lesions, each with a halo of erythema. Seven of these lesions were arranged in a linear fashion on the extensor surface of the right forearm and arm (Fig. 1). One lesion was on the flexor surface of the right forearm. A thin, seropurulent material exuded from the lesions. There was no regional lymphadenopathy. No other abnormalities were present on physical examination.

The temperature was 99°F. by mouth.

Mycologic culture of the lesions (on Sabouraud's medium) revealed a pure growth of *S. schenckii* (Fig. 2). Bacteriologic culture disclosed only a sparse growth of *Staphylococcus albus*.

Examination of the peripheral blood and routine urinalysis were within normal limits. The erythrocyte sedimentation rate was 34 mm. per hour (normal 0 to 20 mm.). The Mazzini flocculation test for syphilis was negative.

A roentgenogram of the chest revealed no significant pulmonary lesions. Agglutinations for brucella and tularemia were negative in all dilutions.

A saturated solution of potassium iodide was given to the patient to take by mouth in increasing doses. Upon reaching the dosage of 30 drops three times a day, after 2½ weeks, she showed intolerance, consisting of malaise, headache, watering of the eyes and "soreness of the teeth." Therapy was continued haphazardly for another week and then discontinued. She was not seen again until 3 months after the original visit, at which time all the lesions were completely healed.

Summary

What is believed to be the first reported case of sporotrichosis in the state of New Hampshire is presented.

To date 8 proved cases of sporotrichosis have been reported in New England.

References

1. Madden, J. F. Sporotrichosis in Minnesota. *Minnesota Med.* **30**: 854, 1947.
2. Gastineau, F. M., Spolyer, L. W., and Haynes, E. Sporotrichosis: report of six cases among florists. *J. A. M. A.* **117**:1074-1077, 1941.
3. Foerster, H. R. Sporotrichosis, occupational dermatosis. *J. A. M. A.* **87**:1605-1609, 1926.
4. Weise, E. C. Prevalence of sporotrichosis in Connecticut: review of three cases and report of new case. *Connecticut M. J.* **6**:841-847, 1942.
5. *Idem.* Sporotrichosis in Connecticut with case report. *New Eng. J. Med.* **205**:951-955, 1931.
6. *Idem.* Another case of sporotrichosis in Connecticut. *New Eng. J. Med.* **211**:353-356, 1934.
7. Blaisdell, J. H. First case of sporotrichosis in New England; certainly first to be reported in literature. *Boston M. & S. J.* **176**:811, 1917.
8. Downing, J. G. Surgical problems of fungous origin: report of case of sporotrichosis in Massachusetts. *New Eng. J. Med.* **212**:1085-1087, 1935.
9. Cragin, D. B., Hardy, T. E., and Shaw, J. F. Sporotrichosis: report of case. *J. Maine M. A.* **9**:93, 1918-1919.

MEDICAL PROGRESS

DIABETES MELLITUS (Concluded)

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BOSTON

Insulin Therapy

The American Diabetes Association has announced the details of the three new standardized insulin syringes that it is recommending for general adoption.[59] These have colored markings corresponding to the strength of the insulin solutions for which they are intended (red, U 40; green, U 80).

Perfection of newer types of insulin and better understanding of the action of the old ones has helped in the control of patients with more severe diabetes. Rohr and Colwell[60] have studied the time-action curves of globin insulin. They showed that in larger doses (60 units) the action of this type of insulin may extend throughout the twenty-four hours. Moreover, regular (soluble) insulin may be mixed with globin, endowing it with a quick action that it lacks. With this information, Sheppe and Sheppe[61] were able to utilize globin or globin-regular mixtures in 50 per cent of their total patients (or 60 per cent of their insulin patients), with fair blood sugar control in most. By using globin in the morning with a second globin or protamine injection at the evening meal, Rabinowitch[62] could attain good blood sugar control even in difficult patients. Thus, globin insulin may deserve wider application than it has hitherto enjoyed.

Mixtures of regular with protamine zinc insulin are still used most widely for the more severe cases. In spite of attempts to simplify their use in the same syringe[63] or by premixing in bottles,[64]† this method still requires rather intelligent co-operation by patients. In an attempt to obtain this combined action more conveniently a stable Danish preparation called "Di-insulin," which is a mixture of regular and twenty-four-hour iso-insulin (phenylureido-insulin) has been used with success.[65] In this country, NPH-50 insulin, not yet commercially available, has been found to yield results similar to a 2:1 mixture.[66, 67] With this insulin, breakfast carbohydrate must be reduced, and extra feedings given at mid-afternoon and bedtime in severe cases. The convenience of NPH-50 insulin is counterbalanced by the fact that in

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†Supplied on a monthly basis (or less).

insulin mixtures the proportions can be adjusted to suit the individual patient.

Any attempt at regulation of patients with severer diabetes requires careful study of the individual case. Even with such attention, a residuum of so-called "brittle" diabetic patients, who cannot be regulated without the inconvenience of repeated injections of regular insulin, remains. Haunz[68] has recently reported his experience with this type of case. It is noteworthy that he had to add a small dose of insulin at 4 to 5 a.m. to counteract the tendency for an early morning rise in blood sugar long known to occur in such severe cases.

Insulin Allergy

Although severe insulin allergy, characterized by generalized reactions, occurs in only about 1 in every 1000 cases, its presence may endanger life and health. In a study of 300 cases of moderate to severe insulin allergy in Sweden, Hult and Jorpes[69] found that such patients could tolerate more highly purified insulin. Some patients required as many as sevenfold recrystallization of the insulin. Moreover, after the use of this insulin for a variable period up to two years, over 90 per cent could then tolerate the amorphous, and this was confirmed by the intracutaneous testing of such patients with regular and sixfold recrystallized insulin.[70] This seems to point to species-specific animal protein rather than the insulin molecule itself as the antigen.* The point has been demonstrated in another way by Lowell,[71] who made 4 out of 24 rabbits insulin resistant by the repeated subcutaneous injection of commercial crystalline insulin. They were not resistant, however, to human insulin. Lowell estimated that the degree of insulin resistance produced in these rabbits was comparable to that of some insulin-resistant human beings. In some of the more severe cases of human insulin resistance (requiring over 1000 units per day for normoglycemic control) Lowell has been able to demonstrate such binding antibodies. However, these represent only a small percentage of all insulin-resistant cases.

Miscellaneous Complications

There is increased interest in the vascular and other degenerative complications of diabetes. Bailey[72] reviewed an adult group of 500 patients and found a statistically valid positive correlation of the incidence of arteriosclerotic heart disease with diabetic control as well as the duration of the disease. Stearns, Schlesinger and Rudy,[73] in an excellent study of coronary-artery disease among diabetic patients, demonstrated significantly increased incidence after forty years of age and after ten years of diabetes. This emphasizes the need for an increased index of suspicion of coronary-artery disease in the clinical handling of such patients. Patients who require a leg amputation have a poor prognosis both for life and health; only 40 per cent survive another three years and of the survivors 40 per cent will require a second amputation.[74]

In diabetic patients who fail to gain weight while on an adequate diet the presence of poor fat absorption with unsuspected steatorrhea should be considered as a possible cause.[75] Among a group of patients with diabetic neuropathy 60 per cent were found to have such gastrointestinal symptoms as cramps, anorexia, vomiting, severe constipation and diarrhea.[76] However, gastrointestinal x-ray films showed a wide variety of abnormalities, none of them characteristic.

The cause for the increased susceptibility to infection in diabetes has never been found. Recent work has shed more light on this problem. Alloxan-diabetic rabbits were found capable of attaining a normal precipitin titer when immunized to purified crystalline egg albumen.[77] The antibody response to typhoid H antigen in a group of 64 diabetic patients was normal in many, regardless of hyperglycemia.[78] It was definitely diminished only in those with hypoalbuminemia. Oral supplements of protein hydrolysates over a thirty-day period corrected both the deficient blood protein level and antibody formation. Pneumococcus Type II grew at an increased rate in whole blood of alloxan-diabetic rabbits as compared with the normal, whereas growth was equal in the respective plasmas.[79] The conclusion was therefore drawn that for Type II pneumococcus the difference in resistance resided in the decreased leukocytic defenses of the diabetic patient.

Bladder abnormalities as revealed by cystoscopy were found in 42 per cent of 82 cases of diabetes studied.[80] These consisted of distortion and diminution in size of the trigone, reduction in length of the posterior urethra and thickening of the vesical neck. In the discussion of this article, Kutzman indicated that he had achieved good results from medical treatment alone in 8 such symptomatic cases. For the more serious cases of diabetic atonic bladder with secondary infection and disability Emmett et al.[81] discussed transurethral resection and gave 3 illustrative cases with good results.

Intercapillary Glomerulosclerosis

The clinical features of this serious complication of diabetes have been brought up to date by the recent review of Rifkin et al.,[82] who emphasized the fact that, as with other diabetic complications, this one may occur insidiously in the course of an undiagnosed and untreated diabetes. Considerable laboratory data for correlation with the clinical features were recorded. The presence of doubly

*At present in the United States most commercial insulin is a mixture of beef and pork, with the exception of that of Sharp and Dohme (beef). Eli Lilly and Company will supply on request a pork or beef crystalline product (special Iletin). Accordingly, since most patients have been sensitized by the mixed products, a highly purified recrystallized insulin would be needed. This is not yet available in the United States. However, Burroughs Wellcome and Company are about to market a more highly purified globin insulin that may be useful.

refractile fatty cells or casts was found of value diagnostically. The edema usually appeared prior to the diminution in serum albumin. Derow and Schlesinger[83] had the unique opportunity of timing the development of this syndrome pathologically in the human diabetic patient. The full pathological picture developed within four years, and the clinical syndrome developed within two years after an operative specimen showed the definite absence of this lesion. The discovery of these renal lesions in alloxan-diabetic rats has further confirmed their high degree of specificity.[84]

Corcoran, Taylor and Page[85] found the changes in renal hemodynamics in this condition to be similar to those of glomerulonephritis. The elevated renal threshold sometimes noted in this disorder was explained by a disproportionately reduced glomerular function relative to tubular function. Nielsen[86] found this also to be the explanation of the high renal threshold of diabetic patients without renal disease.*

Retinitis

The pathology of diabetic retinitis has recently been clarified by the application of a new carbohydrate stain.[87, 88] This clearly outlines the basement membrane throughout the entire retinal vascular tree. The capillary aneurysms seen in diabetic retinas were shown to have "both an afferent and an efferent connection and to be, therefore, true aneurysmal dilatations, not endothelialized petechiae," as hitherto imagined. They are situated in the inner nuclear layer of the retina and the frank hemorrhages are frequently adjacent to them. Ophthalmoscopically, the aneurysms would be seen as "petechial hemorrhages," and the subsequent rupture noted as the frank hemorrhages.

Clinically, the pathogenesis of this complication has been linked with increased capillary fragility and the Kimmelstiel–Wilson syndrome.[89-91] The development of all three have their closest relation to the duration of the diabetes. They have, however, been shown to occur independently of one another, indicating a parallel development. The pathological technic described above is being extended to the kidneys, in which there is also considered to be a degenerative weakening of the walls of the capillaries.

Therapeutically, the effect of rutin, up to 300 mg. per day, has been adequately explored by several investigators. The irregular and uncertain course of retinitis is hard to evaluate and might easily encourage uncritical conclusions. Even with the newer pathological concept described above, a drug that would prevent hemorrhage subsequent to the aneurysmal-petechial stage would be welcome. Unfortunately, the results of treatment have not been encouraging to date, even though the skin capillary fragility may have been corrected.[90, 91]

Surgery

Diabetic patients undergoing surgery frequently have had a preceding period of poor regulation with attendant acetonuria, negative nitrogen balance and depletion of liver glycogen. Maloney et al.[92] have shown that ether anesthesia by itself results in further liver glycogen depletion. This could be minimized in large measure by a high-carbohydrate diet or intravenous administration of glucose preoperatively.

The inability of nondiabetic patients to assimilate and utilize glucose during anesthesia and surgery was studied by Pareira et al.,[93, 94] who showed that the technic and rate of infusion are important in avoidance of urinary loss: a 10 per cent solution should be given at a rate of 10 gm. (100 cc.) per hour during the first half hour, and then accelerated to a level of 45 gm. (450 cc.) per hour for the remainder. They administered 1000 cc. of 10 per cent glucose in this manner to 24 patients before operation, starting at the onset of anesthesia. All patients retained 84 per cent or more of the glucose and the usual postoperative ketonuria was reduced to a minimum.[94] The authors advocated over 200 gm. of glucose a day in 10 per cent solution as optimum for this purpose. These studies in the normal subject apply with even greater emphasis to the diabetic patient undergoing surgery, for the latter frequently starts with a preoperative handicap of diminished liver glycogen and ketonuria.

In the actual management of the surgical patient with diabetes Alexander and his associates[95] illustrated a six-hour alimentation method that is theoretically excellent. This would be difficult to manage in the average hospital, which is not set up to serve meals at 3 a.m. However, a modification employing the usual three meals and a 10 p.m. feeding might serve as a substitute.

Pregnancy

The problem of preventing late fetal mortality in diabetic pregnancy has been well presented in an editorial review in the *British Medical Journal*.[96] Control of the diabetes is not the decisive factor in the complete prevention of this complication. Speculation regarding the cause of the high fetal mortality in diabetic pregnancies must include the fact that it may be increased for many years prior to diagnosis of diabetes. Barns et al.[97] have investigated the role of the anterior pituitary hormones, growth or diabetogenic, in this phenomenon. They found that an anterior pituitary extract administered to diabetic pregnant rats increased the stillbirth rate from 33 to 100 per cent.

White[98] has followed a large group of pregnant diabetic women and believes that there was a definite relation between fetal mortality and the

*This elevation of renal threshold may develop in undiagnosed diabetes, thus causing a delay in both the glycosuria and the diagnosis itself.

presence in the mother of various diabetic degenerative complications such as retinitis, hypertension, albuminuria and calcified pelvic arteries. She divided the patients into several definite prognostic categories dependent upon the presence of these complications together with the duration of the diabetes. She found hormonal imbalance in 90 per cent of all her patients, and in 97 per cent of the patients with fetal loss. She believed that the correction of these abnormalities by hormonal therapy (stilbestrol and progesterone) had favorably influenced fetal survival, increasing it from 58 to 89 per cent. However, the groups were not controlled so as to distinguish between the influence of the degenerative and the hormonal factors. Yet the evidence is strongly suggestive that hormonal imbalance is at least associated with unfavorable results and justifies routine hormonal therapy at present. For this purpose, one may use the relatively convenient schedule of oral administration of diethylstilbestrol proposed by Smith.[99] The more fundamental degenerative element still presents a therapeutic challenge.

With or without hormonal therapy large, unhealthy babies result. It is important to recognize that the neonatal mortality stems mainly from the presence of weak respiratory function and overhydration. Gellis et al.[100] have presented an outline of therapy serving to minimize the effect of these factors after delivery.

Coma

More complete studies of metabolic balance have recently contributed valuable information on this subject. One can now more closely estimate the deficits that have occurred in the body economy in the average case: 26 gm. of sodium chloride, 10 per cent of body weight of water, approximately 5 gm. of phosphorus[101] and 3 to 16 gm. of potassium.[102] An initially normal or even high serum potassium does not preclude a tissue deficit, which is the more important with relation to the symptoms of potassium deficiency that may be fatal. This state can be adequately followed, and correction confirmed by electrocardiography.[103] A critical drop in the blood potassium concentration occurs early in the therapy of diabetic coma. It is due to diuresis, dilution by potassium-free fluids administered (regardless of type) and entry of potassium into cells associated with improved glucose utilization.[101, 105]

By the mechanism of "dilution acidosis" large amounts of sodium chloride and water may also further aggravate the acidosis.[106] This effect may be avoided in part by replacing a portion of the sodium chloride by sodium bicarbonate in therapeutic solutions.

Cerebral metabolism and blood flow have been studied by new technics.[107] A reduction of cerebral oxygen consumption below 40 per cent of normal was associated with poor response to therapy and death. This metabolic change in the brain was best correlated with blood ketone levels rather than blood carbon dioxide or cerebral blood flow.

It has further been shown that the administration of glucose in water in acidosis increases elimination of sodium chloride and water even more than potassium.[108, 109] In a well studied group of patients in acidosis Franks et al.[109] demonstrated that early glucose administration, even with saline solution, decreased the rate of recovery, encouraged excessive diuresis and tissue dehydration, and did not increase glucose metabolism or correct the acidosis sooner.[109] However, the addition of phosphate not only replaced the deficit but also speeded up the recovery of normal blood carbon dioxide levels and of consciousness.[110]

From a variety of such studies certain modifications may be introduced into present-day coma routines. Initially (first five or six hours) more hypotonic solutions containing more sodium than chloride (in a ratio of 1.3:1) should be used than hitherto. This may be accomplished by replacement of part of the sodium chloride by sodium lactate (or bicarbonate) and the addition of water to physiologic saline solution.[111] Glucose is best avoided until after this initial phase, and then administered intravenously with phosphate and potassium. This is an average and should be modified by the individual needs as revealed by the duration of coma, amount of vomiting and laboratory studies. As an example, although glucose administration was shown to be undesirable in the cases reported by Franks et al.,[109] all, without exception, had high initial blood sugar values. Boulin[112] has shown that cases of unmistakable coma occur (19 out of 81 cases) with blood sugar values below 300 mg. per 100 cc.[112] In such cases, to avoid hypoglycemia while the acidosis is being corrected with large doses of insulin, glucose would have to be administered relatively early.

The correction of biochemical defects still leaves a significant mortality from complications, among which cardiovascular collapse and renal failure seem to be potentially amenable to treatment. Study of the low and irreversible blood pressure in diabetic coma has shown that it is due to a greatly decreased total peripheral resistance since cardiac output is not reduced.[113] Restoration of the carbon dioxide combining power and administration of Methedrine, Pitressin and digitalis were not found helpful, whereas large intravenous, nonprotein infusions tended to cause pulmonary edema and a falling oxygen saturation. Renal failure due to lower-nephron nephrosis may occur, perhaps as a result of prolonged shock. Two such cases have been described, 1 of which responded only temporarily to transperitoneal lavage.[114]

References

59. Palmer, L. J., and Peck, F. B. Official syringe of American Diabetes Association. *J. A. M. A.* **139**:7-9, 1949.
60. Rohr, J. H., and Colwell, A. R. Comparative time action of globin insulins. *Arch. Int. Med.* **82**:54-62, 1948.
61. Sheppe, W. M., and Sheppe, W. M., Jr. Evaluation of globin insulin in treatment of diabetes mellitus. *West Virginia M. J.* **45**:79-83, 1949.
62. Rabinowitch, I. M., Fowler, A. F., Gordon, A. L., and Mountford, M. Globin insulin. *Canad. M. A. J.* **56**:595-605, 1947.
63. Muntz, H. H. Method for mixing insulins. *J. A. M. A.* **141**:987, 1949.
64. Wiesel, B. H., Riser, A. B., and Kahn, S. S. Insulin mixtures: experiences in use of extemporaneous bottle mixtures in diabetic clinic patients. *Am. J. Med.* **7**:655-659, 1949.
65. Brull, L., et al. Essai clinique de la diinsuline. *Rev. méd. Liège* **2**: 629-639, 1947.
66. Gabriele, A. J., and Marble, A. Clinical experience with new modified protamine insulin (NPH-50). *Am. J. Digest. Dis.* **16**:197-206, 1949.
67. Kirkpatrick, N. R. Experiences with new insulin. *Proc. Staff Meet., Mayo Clin.* **24**:365-370, 1949.
68. Haunz, E. A. Approach to problem of "brittle" diabetic patient: study of six cases. *J. A. M. A.* **142**:168-173, 1950.
69. Hult, H., and Jorpes, E. Is insulin antigenic? *Lancet* **1**:780, 1949.
70. Paley, R. G. Mechanism of cutaneous reactions to insulin. *Lancet* **2**:1216, 1949.
71. Lowell, F. C., and Franklin, W. Induced insulin resistance in rabbit. *J. Clin. Investigation* **28**:199-206, 1949.
72. Bailey, R. L., Jr. Arteriosclerotic heart disease in diabetics. *Virginia M. Monthly* **76**:411-415, 1949.
73. Stearns, S., Schlesinger, M. J., and Rudy, A. Incidence and clinical significance of coronary artery disease in diabetes mellitus. *Arch. Int. Med.* **80**:463-474, 1947.
74. Silbert, S. Mid-leg amputations for gangrene in diabetic. *Ann. Surg.* **127**:503-512, 1948.
75. Field, H. Jr., Klendshoj, N. C., and Bowen, B. D. Steatorrhea in diabetes mellitus. *Am. J. Digest. Dis.* **15**:298-301, 1948.
76. Hodges, F. J., Rundles, R. W. and Hanelin, J. Roentgenologic study of small intestine. II. Dysfunction associated with neurologic diseases. *Radiology* **49**:659-673, 1947.
77. Payne, T. P. B., and Cruickshank, A. H. Antibody formation in alloxan diabetes: comparison of precipitin response to egg albumen of normal rabbits and of rabbits with alloxan diabetes. *Bull. Johns Hopkins Hosp.* **83**:326-329, 1948.
78. Wohl, M. G., Waife, S. O., Green, S., and Clough, G. B. Relationship of blood sugar and hypoproteinemia to antibody response in diabetics. *Proc. Soc. Exper. Biol. & Med.* **70**:305-307, 1949.
79. Cruickshank, A. H., and Payne, T. P. B. Anti-pneumococcal powers of blood in alloxan-diabetes in rabbit. *Bull. Johns Hopkins Hosp.* **84**:334-343, 1949.
80. Lich, R., Jr., and Grant, O. Vesical abnormalities incident to diabetes mellitus: *J. Urol.* **59**:863-874, 1948.
81. Emmett, J. L., Daut, R. V., and Sprague, R. G. Transurethral resection for neurogenic vesical dysfunction in cases of diabetic neuropathy. *J. Urol.* **61**:244-257, 1949.
82. Rifkin, H., Parker, J. G., Polin, E. B., Berkman, J. I., and Spiro, D. Diabetic glomerulosclerosis: clinical and pathologic observations with special reference to doubly refractile fatty cells and casts in urine. *Medicine* **27**:429-457, 1948.
83. Derow, H. A., and Schlesinger, M. J. Evolution of intercapillary glomerulosclerosis. *New Eng. J. Med.* **241**:7-12, 1949.
84. Foreign Letters: Buenos Aires: renal lesions of experimental diabetes. *J. A. M. A.* **140**:425, 1949.
85. Corcoran, A. C., Taylor, R. D., and Page, I. H. Functional patterns in renal disease. *Ann. Int. Med.* **28**:560-582, 1948.
86. Nielsen A. L. On mechanism of glycosuria. I. *Acta med. Scandinav.* **130**:219-231, 1948.
87. Friedenwald, J. S. New approach to some problems of retinal vascular disease. *Am. J. Ophth.* **32**:487-498, 1949.
88. Ashton, N. Vascular changes in diabetes with particular reference to retinal vessels. *Brit. J. Ophth.* **33**:407-420, 1949.
89. Croom, J. H., and Scott, G. I. Retinal and vascular damage in long-standing diabetes. *Lancet* **1**:555-558, 1949.
90. Peck, F. B., and Mann, M. Effect of hesperidin methyl chalcone (vitamin P), on diabetic retinopathy. *Am. J. M. Sc.* **217**:277-282, 1949.
91. Beardwood, J. T., Roberts, E. and Trueman, R. Observations on effect of rutin and hesperidin in diabetic retinitis. *Proc. Am. Diabetes A.* **8**:243-256, 1948.
92. Maloney, A. H., Graves, B. Jr., and Rhodes, L. C., Metabolic changes in anesthesia under various states of nutrition. *J. Nat. M. A.* **41**:202-214, 1949.
93. Pareira, M. D., and Somogyi, M. Rationale of parenteral glucose feeding in postoperative state. *Ann. Surg.* **127**:417-425, 1948.
94. Pareira, M. D., and Probstein, J. G. Glucose assimilation during anesthesia and surgery. *Ann. Surg.* **129**:463-467, 1949.
95. Alexander, J. B., Loomis, A. H., and Lee, C. T., Jr. Management of diabetes mellitus during acute complications and surgery. *M. Clin. North America* **33**:1413-1419, 1949.
96. Editorial. Diabetes and pregnancy. *Brit. M. J.* **1**:62, 1949.
97. Barns, H. H. F., Lindan, O., Morgans, M. E., and Swyer, G. I. M. Experimental diabetic pregnancy; effect of alloxan and anterior-pituitary extract on pregnant rat. *J. Endocrinol.* **6**:xvi, 1949.
98. White, P. Pregnancy complicating diabetes. *Am. J. Med.* **7**:609-616, 1949.
99. Smith, O. W., Diethylstilbestrol in prevention and treatment of complications of pregnancy. *Am. J. Obst. & Gynec.* **56**:821-834, 1948.
100. Gellis, S., White, P., and Pfeffer, W. Gastric suction: proposed additional technic for prevention of asphyxia in infants delivered by cesarean section: preliminary report. *New Eng. J. Med.* **240**: 533-537, 1949.
101. Butler, A. M., Talbot, N. B., Burnett, C. H., Stanbury, J. B., and MacLachlan, E. A. Metabolic studies in diabetic coma. *Tr. A. Am. Physicians* **60**:102-109, 1947.
102. Danowski, T. S., Peters, J. H., Rathbun, J. C., Quashnock, J. M., and Greenman, L. Studies in diabetic acidosis and coma, with particular emphasis on retention of administered potassium. *J. Clin. Investigation* **28**:1-9, 1949.
103. Nadler, C. S., Bellet, S. and Lanning, M., Influence of serum potassium and other electrolytes on electrocardiogram in diabetic acidosis. *Am. J. Med.* **5**:838-848, 1948.
104. Greenman, L., Mateer, F. M., Gow, R. C., Peters, J. H., and Danowski, T. S. Some observations on development of hypokaliemia during therapy of diabetic acidosis in juvenile and young adult subjects. *J. Clin. Investigation* **28**:409-414, 1949.
105. Nadler, C. S., Bellet, S., Reinhold, J. G., and Lanning, M. Alterations in serum potassium and their relation to certain constituents of blood in diabetic acidosis. *Am. J. M. Sc.* **218**:308-317, 1949.
106. Shirey, G. T., and Holman, J. Dilution acidosis. *Ann. Int. Med.* **28**:557-559, 1948.
107. Kety, S. S., Polis, B. D., Nadler, C. S., and Schmidt, C. F. Cerebral blood flow and metabolism in diabetic acidosis and coma. *J. Clin. Investigation* **26**:1186, 1947.
108. Brodsky, W. A., and Rapoport, S. Renal osmotic work during forced diuresis in dehydration in man. Effect of glucose urea loading. *J. Clin. Investigation* **27**:526, 1948.
109. Franks, M., Berris, R. F., Kaplan, N. O., and Myers, G. B. Metabolic studies in diabetic acidosis. I. Effect of early administration of dextrose. *Arch. Int. Med.* **80**:739-762, 1947.
110. *Idem.* Metabolic studies in diabetic acidosis. II. Effect of administration of sodium phosphate. *Arch. Int. Med.* **81**:42-55, 1948.
111. Butler, A. M. Parenteral therapy in diabetic coma. *Pediatrics* **2**: 701, 1948.
112. Boulin, R. La glycémie dans le coma diabétique. *Presse méd.* **56**: 753, 1948.
113. Howarth, S., McMichael, J., and Sharpey-Schafer, E. P. Low blood pressure in diabetic coma. *Clin. Sc.* **6**:247-255, 1948.
114. Brody, D. A. Lower nephron syndrome in association with diabetic coma: report of two cases. *Memphis M. J.* **23**:71, 1948.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 36301

PRESENTATION OF CASE

A sixty-five-year-old man entered the hospital complaining of constipation and abdominal pain.

The patient had always considered himself well and did heavy daily labor, lifting 50-pound cans without difficulty of any sort. Two and a half years prior to admission he noted mild abdominal discomfort and increased flatus. A gastrointestinal series taken at another hospital revealed a prepyloric gastric ulcer and evidence of gastroduodenitis. A barium enema showed no organic disease in the large bowel. A mild but unspecified level of hypertension was discovered. The symptoms cleared rapidly under dietary treatment. Three weeks before admission the patient slipped on an icy pavement and fell, slightly bruising his left hip. Up to this point he had had normal daily bowel movements, but following the accident he noted obstinate constipation, producing only a few small marbles of feces with the aid of laxatives or enemas. However, he continued to pass large quantities of gas by rectum throughout his illness. A few days after the onset of constipation, he began to have severe, cramping, nonradiating lower midabdominal pain that started twenty or thirty minutes after eating and was relieved by an enema or bowel movement. He became anorexic because of this pain but had no nausea or vomiting. There was a 20-pound weight loss within the following three weeks. Five days before admission the patient entered another hospital, where a sigmoidoscopic examination was reported as negative. A barium enema was interpreted as consistent with partial, probably intermittent volvulus of the lower descending colon and showed dilatation of the colonic flexures and some small-bowel loops. There was no evidence of intrinsic disease. Fluoroscopy revealed a pulsatile mass in the left lower quadrant adjacent to but not arising from the sigmoid. The thoracic aorta was also noted to be dilated. The patient was then referred to this hospital.

The patient had a chronic cough of ten years' duration, which of late months had been productive of small amounts of faintly blood-streaked sputum. He had occasionally noticed palpitations and a heaving precordium while lying on his back, but had no other cardiac symptoms. He had nocturia (two times) of long standing. All signs and symptoms of syphilis were specifically denied. His physician reported that the heart rate had been regular up to three days before entry.

Physical examination revealed a well nourished, well developed man in no distress. The trachea was in the midline, and the neck veins were flat. The left posterior chest was dull to percussion, with decreased breath sounds and decreased vocal and tactile fremitus. Coarse rhonchi were heard throughout, being more plentiful on the left. The left border of cardiac dullness and point of maximum impact were at the anterior axillary line, and a strong precordial heave was noted. The heart was fibrillating at a rate of 160, with a pulse deficit of 80. The heart sounds had a tick-tock quality at the apex. No murmurs were heard. The radial pulses were equal, and the femoral and popliteal pulses were easily felt bilaterally. The posterior-tibial and dorsalis-pedis pulses were absent. A rounded, slightly tender pulsatile mass was felt in the left lower quadrant that appeared to extend in the midline into the upper abdomen. Peristaltic sounds were active and normal. Rectal examination revealed quantities of hard, dry feces but no intrinsic abnormalities. Neurologic examination was negative.

The temperature was 98°F., the pulse 80 and grossly irregular, and the respirations 24. The blood pressure was 160 systolic, 95 diastolic.

The urine gave a green sugar reaction, but was otherwise normal. Examination of the blood demonstrated a hemoglobin of 13.5 gm. per 100 cc. and a white-cell count of 15,000, with 91 per cent neutrophils. A blood Hinton test was negative. Two stool specimens were guaiac negative. An electrocardiogram showed auricular fibrillation at a rate of 180, with an uncontrolled ventricular rate.

X-ray films of the chest (Fig. 1) and fluoroscopy revealed a pulsatile mass in the posterior mediastinum that appeared to be within the left lung and extended along the left cardiac border lateral to the aortic knob, causing deviation of the esophagus anteriorly.

The left main bronchus was elevated by the mass and the left-lower-lobe bronchus was not visualized. The left lower lobe was partially collapsed. The heart appeared to be enlarged, and the aorta to be tortuous and markedly widened. The left costophrenic angle was obliterated. The

seventh dorsal vertebra was partially collapsed, but there was no obvious bone erosion in any of the vertebral bodies. Plain films of the abdomen showed a colon filled with gas and fecal material. There was no appreciable amount of air in the small intestine. The liver was somewhat enlarged, but the spleen was of normal size. No masses were recognized in the lower abdomen.

The patient received digitoxin and digoxin on the first four hospital days, and on the fifth and sixth he received quinidine in addition. The apical pulse rate fell to 120 under treatment and, after

FIGURE 1.

quinidine was started, there was a return of normal sinus rhythm. Throughout his hospital course the patient complained of severe abdominal pain, intensified by eating, and only partially relieved by enemas, morphine and Demerol. On the fifth day he had three small hemoptyses of approximately half a teaspoonful of blood each, and the temperature rose to 101°F. Late in the evening of the sixth day he complained of slight respiratory difficulty, lapsed quickly into coma and died half an hour later.

DIFFERENTIAL DIAGNOSIS

DR. RICHARD J. CLARK*: Dr. Wyman, may we see the x-ray films?

DR. STANLEY M. WYMAN: These x-ray films were all taken within a period of twenty-four hours and show, through the heart shadow, a dense homogeneous soft-tissue mass, which is rather fusiform in character and conforms to the course and location of the descending thoracic aorta. It is seen on the lateral view lying posteriorly. The posterior border of the mass, however, cannot be seen. There is fluid in the left pleural space. There appear to be increased markings in the left-lower-lung fields, suggesting atelectasis of the left lower lobe. According to the fluoroscopist, this mass within the heart shadow was seen to pulsate at a different time interval from the heart itself but to follow the same rate as the heart. The film of the abdomen reveals no abnormal soft-tissue masses and no unusual gas pattern.

DR. CLARK: What about the bone erosion that was mentioned?

DR. WYMAN: There is slight collapse of one of the midthoracic vertebral bodies that does not appear to be the result of actual bone destruction, and I am unable to say much more about it.

DR. CLARK: It does not look particularly like a metastasis does it?

DR. WYMAN: No; it does not.

DR. CLARK: What is your batting average on visualizing aneurysms in the abdominal cavity?

DR. WYMAN: I think we see most of the calcified abdominal aneurysms — a good many anyway.

DR. CLARK: There are four major problems to be solved: the first is the lesion in the mediastinum or lung; the second is the abdominal disease, the third is the cardiac lesion; and the fourth is the terminal event. First I would like to dispose of what I am going to assume is a "red herring" — namely, the prepyloric ulcer. Everyone is aware that prepyloric ulcers may well be malignant. Could that have had anything to do with the subsequent sequence of events? The symptoms cleared, and there was no progressive weight loss, no deterioration up to the sudden events just before entry, and no anemia. There was a negative guaiac reaction on the stool, and there were no evidences of metastases to be found on x-ray examination. I am going to relegate the ulcer to ancient history.

Now we come to the question of the mediastinum. At the onset I am going to assume on the basis of evidence presented, particularly on the basis of the x-ray films, that this patient had an aneurysm of the descending arch of the aorta. For ten years he had a cough and more recently he had been raising some blood-stained sputum. On physical and x-ray examination at the time of entry there was evidence of bronchial obstruction, collapse of the left lower lobe and some fluid in this region. Did he possibly have a bronchogenic carcinoma? Certainly, a bronchogenic carcinoma could produce all these findings exactly as described. However, there was no definite proof of this, and I see no means of making this diagnosis. We know, on the other hand, that aneurysms can very well cause pressure on the bronchi, can cause bronchial obstruction, can even erode through bronchi and can produce all this picture, including the raising of small amounts of blood-tinged sputum. So that

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without further ado, I shall assume that the findings in the chest were all on the basis of this thoracic aneurysm with pressure on the bronchus producing the signs and symptoms as recorded.

One might ask if the chest fluid could have been due to cardiac failure. I would rule this out on the basis of the unilateral fluid. Fluid on the left side alone from congestive heart failure is unusual unless there is some process obliterating the pleural space on the right and we have no evidence of such a process in this particular case.

All was apparently well until the patient's fall three weeks before entry, following which he developed the abdominal pain and then became suddenly constipated. He presented all the signs and symptoms of a partial intestinal obstruction, and yet studies failed to reveal any intrinsic bowel disease and there was a negative guaiac reaction. A pulsatile mass in the left lower quadrant was seen on fluoroscopy in the other hospital and felt in this hospital. It was also reported to be tender. I do not believe that this patient had any primary intrinsic bowel lesion. What about the suggestion of a volvulus, which was seen in the other hospital? I recently heard of a woman who suddenly developed a syndrome very similar to this and who at autopsy had an aneurysm of the abdominal aorta that had begun to leak into the retroperitoneal space; the collection of blood in the retroperitoneal space had actually produced a kinking and pushing up of the sigmoid. In spite of the negative findings on the plain film at this hospital, from the descriptions given I can only consider that the patient under discussion had a second aneurysm in the abdomen. I believe it likely that at the time of his fall there was a partial rupture with leakage. Such a rupture may occur, and the patient may still live for some time thereafter. There was probably retroperitoneal bleeding, with resultant pressure and distortion of the bowel, and this could also explain the leukocytosis on entry. I wondered at first if this patient had actual dissection of the abdominal aorta. This would seem unlikely, however, because there was not enough pain and there was no disturbance of the circulation in the femoral or posterior-tibial arteries.

We come next to the question of where this abdominal aneurysm arose. Was it aortic or was it iliac? From the description of the physical examination, I believe it would fit more properly as an aortic aneurysm. If it were of the iliac artery, I would expect a disturbance of pulsation in the left femoral or left popliteal artery, which the patient did not have.

If we are to assume that this patient had two aneurysms, one of the descending thoracic aorta and one of the abdominal aorta, what was the etiology? Statistics show that thoracic aneurysms are twenty times more commonly syphilitic than arteriosclerotic whereas abdominal aneurysms are about nine times more commonly arteriosclerotic than syphilitic. "You pay your money and take your choice." This man denied all stigmas and symptoms of syphilis; he had a negative blood test, but this does not necessarily rule out syphilis. The etiology in this case is speculative. I would be a little bit happier about making a diagnosis of arteriosclerotic aneurysm if sclerosis had been seen on x-ray study in either of them. I think the greater probability is that they were arteriosclerotic in origin.

What about this cardiac lesion? The chief evidence of cardiac disease is marked enlargement both on x-ray and physical examination, in addition to very rapid auricular fibrillation. The patient had little cardiac symptomatology. I would be interested in knowing if there was any further description of his heart. The description given in the protocol was when he was fibrillating at a rate of 160 to 180. Certainly, murmurs can be very easily missed in the presence of any such cardiac rate. When the rate slowed, he may have had murmurs that were not described. The fact that the blood pressure was 160 systolic, 95 diastolic, clearly rules out aortic regurgitation. I see no evidence for valvular heart disease. I have seen the electrocardiogram, and it adds little to what is recorded in the protocol; the QRS complexes were normal; the T waves were not remarkable and gave no definite evidence of any coronary-artery disease. Therefore, I shall fall back on the assumption that this patient had ventricular hypertrophy on the basis of hypertension that had been present for some time. Aneurysms in themselves as a rule do not cause cardiac disease unless they involve the aortic valve or the coronary ostia. The patient did have dilatation of the entire aorta, and it is possible that he had some asymptomatic coronary-artery obstruction. The extremely rapid fibrillation requiring massive doses of quinidine is a little bit disquieting. On the other hand the patient had been fibrillating for only a few days and was converted to normal rhythm fairly quickly, which would be against a long-standing lesion such as mitral stenosis. The question of why he should fibrillate so rapidly comes up. Pulmonary infarction is one of the things that will cause such a very rapid rate with fibrillation and brings me to the discussion of the terminal event.

The patient had spat up some blood, and he may have had pulmonary embolism. On the other hand, the respiratory difficulty could all be explained on other bases. Further bleeding as the result of bronchial erosion from the aneurysm is very possible. Could a thoracic aneurysm have ruptured? This, I think, is entirely unlikely inasmuch as the terminal events were not sufficiently dramatic or sufficiently rapid. He could have had some further leakage from the abdominal aneurysm, giving him some anemia toward the end and a

downhill course. He could have developed a bronchopneumonia or some degree of congestive heart failure. Any of these things could have happened at the end, and I am not at all certain which one of them precipitated death.

A PHYSICIAN: Regarding the final event, I wonder if this case is one that is always talked about and never seen — a patient who has embolism following the return of fibrillation to normal with quinidine.

DR. CLARK: I would consider this unlikely because the patient had been fibrillating for such a short time. It is thought by some to be a hazard of quinidine therapy but especially in patients who have been fibrillating for some time.

DR. TRACY B. MALLORY: Dr. Pittman, have you anything to add?

DR. HELEN B. Pittman: I think Dr. Clark has gone over the same ideas that we went over. There were no murmurs in the heart when it slowed down, and I believe that we thought the aneurysm was probably iliac rather than aortic.

DR. CLARK: Why did you think that?

DR. PITTMAN: Just because the mass was so far out on the side and seemed to coincide with the angle of the iliac artery.

CLINICAL DIAGNOSES

Arteriosclerotic aneurysms, ruptured, of thoracic aorta and iliac artery.
Arteriosclerotic heart disease, with auricular fibrillation.
Intermittent bowel obstruction due to compression by abdominal aneurysm.

DR. CLARK'S DIAGNOSES

Arteriosclerotic aneurysms of descending thoracic and abdominal aorta, with slow rupture of latter.
Hypertensive heart disease.
? Pulmonary embolism, ? bronchopneumonia.

ANATOMICAL DIAGNOSES

Arteriosclerotic aneurysms of both iliac arteries and abdominal and descending thoracic aorta.
Slow rupture of thoracic aneurysm, with mediastinal hemorrhage.
Cardiac hypertrophy, hypertensive type.
Bronchopneumonia.
Gastric ulcer, prepyloric, active.

PATHOLOGICAL DISCUSSION

DR. MALLORY: I cannot settle the question whether the pulsating mass was an aortic or iliac aneurysm because he had both. He had aneurysms of both iliac arteries, a separate one of the abdominal aorta with a fourth much larger aneurysm of the descending thoracic aorta. There was no evidence of retroperitoneal hemorrhage around any of the three intra-abdominal aneurysms, but there was evidence that the thoracic aneurysm had leaked although it had not ruptured dramatically. There was considerable extravasation of blood into the left lower lobe of the lung, which could not be dissected away from the wall of the aneurysmal sac. There was some dissection of blood along the outer layers of the esophageal wall and down on the stomach wall, where a hematoma of considerable size was likewise found.

Regarding the other phenomena, there was evidence of a considerable degree of acute infection in the lung as well as hemorrhages so the two processes were superimposed and made a confusing picture. It would be difficult to tell which had occurred first. There unquestionably was pneumonia, and it looked as if this were progressing in places to abscess formation. There was a still active prepyloric ulcer, but sections through its wall showed no malignant degeneration. It was a perfectly benign peptic ulcer. The heart was hypertrophied, weighing 430 gm. There was no valvular heart disease, and the coronary arteries were wide and capacious. The vessels in various organs and particularly in the kidneys showed the hyaline arteriolar sclerosis of long-standing benign hypertension, and I think that we must assume that the cardiac disease was secondary to long-standing hypertension.

DR. ALFRED E. KRANES: Were they arteriosclerotic aneurysms?

DR. MALLORY: That, I think, remains a question that cannot be entirely settled one way or the other. The facts that there were four of them, that the iliac arteries were involved and that there was histologically a considerable amount of calcification in the walls, even though it did not show up on x-ray examination, are points in favor of arteriosclerotic aneurysms. There was, however, focal destruction of the media, and there were many lymphocytes in the adventitia and I can imagine that some pathologists would want to call them syphilitic. My personal opinion is that they were probably arteriosclerotic. I am sure that lymphocytes in the adventitia of the aorta are inadequate evidence of syphilis.

CASE 36302

PRESENTATION OF CASE

A forty-nine-year-old wool sorter entered the hospital complaining of right-upper-quadrant pain.

The patient had been in relatively good health despite a heavy alcoholic history until approximately four months prior to entry, when he became aware of a steady constant pain in the right upper quadrant unrelated to meals but aggravated by lying on the right side. His appetite remained good, and he continued to work. Associated with this he noticed a general lightness of the abdomen,

and his trousers began to feel tight. One month before entry the pain shifted into the midepigastrium, and at this time he ceased drinking alcohol. Ten days before entry he noted a temperature of 101°F., lost his appetite and noticed that the urine was dark and the stools light. He was given sulfonamides for two days, which seemed to have no effect, and was switched to penicillin, with concomitant subsidence of the fever. Antacids were given also but failed to affect the epigastric pain. One week before entry he noted a yellowish color of his skin.

The patient stated that despite the heavy alcoholic intake his appetite had always been good until ten days before entry. He had never previously noticed dark urine, jaundice, hematemesis or melena.

Physical examination showed a well developed and well nourished, chronically ill man in no acute distress. The skin and scleras were markedly icteric. There was no evidence of spider angiomas or petechiae. The pharynx was reddened. The heart and lungs were normal. The abdomen was slightly distended, without tenderness or spasm. The liver was tremendously enlarged; the upper border was percussed at the level of the nipple, and the lower extended from the iliac crest below the umbilicus to the left anterior axillary line. The edge was firm and rounded; no irregularity was felt on the edge or on the surface of the liver. Definite evidence of peritoneal fluid was not observed. No other organs or masses were palpated. Rectal examination showed small external hemorrhoidal tabs. The prostate was slightly enlarged, symmetrical and not unusually indurated.

The temperature was 100°F., the pulse 92, and the respirations 20. The blood pressure was 150 systolic, 96 diastolic.

The urine was dark with a specific gravity of 1.020 and a positive foam test for bile. It contained no albumin nor sugar. The sediment contained 5 to 10 red blood cells per high-power field and rare hyaline and granular casts. The hemoglobin was 15.5 gm. per 100 cc., and the white-cell count 11,000, with 95 per cent neutrophils. A stool was clay colored and guaiac positive. The total protein was 6.6 gm. per 100 cc., with an albumin of 3.7 gm. and a globulin of 2.9 gm. per 100 cc. The alkaline phosphatase was 44.4 units per 100 cc., the serum van den Bergh 9.4 mg. per 100 cc. direct and 13.2 mg. total, and the prothrombin time 65 seconds (normal, 18 seconds). The cephalin flocculation was ++ in twenty-four and +++ in forty-eight hours. The nonprotein nitrogen was 34 mg., the fasting blood sugar 83 mg., the amylase 20 units, and the lipase 1.5 units per 100 cc.

Forty-eight hours after admission the patient became decidedly worse. He was drowsy, euphoric and sweaty and had a rapid pulse. Jaundice was more marked, and there was some tenderness over the liver. He gradually lapsed into deepening coma, and died on the fourth hospital day.

Differential Diagnosis

Dr. Perry J. Culver*: First I would like to say that this man should have had alcoholic cirrhosis of the liver, but I do not believe that he had it. I shall state the reasons for that opinion when I discuss the physical and laboratory data. The history of heavy alcoholic intake is of course very much in keeping with that of the typical patient with alcoholic cirrhosis. The history states that the abdomen gradually became larger and that jaundice appeared. These two symptoms very often will bring such a patient to the doctor, so that from the history itself it looks as if this were a typical case of alcoholic cirrhosis. It is interesting that the patient had a good appetite in spite of heavy drinking. Recently a paper was read by Dr. T. C. Chalmers at the meeting of the Boston Gastroenterology Society with the title "You Have to Eat to Get Cirrhosis." The fundamental idea was that the spree drinkers, who drink heavily without eating, could not continue drinking long enough to damage the liver, but had to recover from their delirium tremens or severe gastrointestinal upsets. Then they would start to eat again, and through remorse or for other reasons would avoid alcohol for a period of time. On the other hand, practically all the cirrhotic patients reported in this paper had eaten only enough while drinking so that the toxic effects of alcohol were not so manifest that they had to stop drinking — ergo, "you have to eat to get cirrhosis." For the sake of those who enjoy a social cocktail, I should like to agree with Dr. Boyd that if one eats well, with adequate protective protein in the diet, alcohol will probably not damage one's liver. If it is true that this man had eaten well, I doubt if alcohol played much of a role in his particular illness.

Next, let us discuss the protocol sentence by sentence or idea by idea. The term "wool sorter" brings up the possibility of anthrax. There was no mention of a skin lesion suggestive of a malignant pustule, nor did the lungs have any lesion in them. I think it was probably coincidental that he was a wool sorter.

The enlargement of the abdomen may have been due to ascites or to a tremendously rapid increase in the size of the liver. In the light of further evidence I choose to believe that it was due to a rapid increase in the size of the liver and not to ascites.

The shift of the pain to the midepigastrium disturbs me a bit. I do not know how to explain it. It is possible that he had a lesion of the gastrointestinal tract such as an ulcer or alcoholic gastritis, but there had been nothing to suggest alcoholic gastritis in the past. I doubt if it was due to

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that. Could it have represented a lesion in the pancreas? One has to consider that very seriously. We know that attacks of acute pancreatitis can be precipitated by acute alcoholism, but why should one have occurred at just this time? Other lesions in the pancreas such as carcinoma still have to be considered seriously. Pain in the epigastrium and in the right upper quadrant can be caused merely by an enlarged, tender liver, which stretches the capsule, and to explain everything for the time being on the basis of liver disease, I should like to think that the midepigastric pain was secondary to the enlargement of the liver.

The loss of appetite ten days before admission was probably due to the rapid progression of the disease and did not represent a causative factor.

The temperature of 100°F., I believe, may be explained on one of three bases: a widespread, active, intrinsic liver disease with extensive necrosis; an obstruction to the biliary drainage somewhat similar to that due to stone or cancer; or a pylephlebitis or secondary type of pyogenic infection of the liver. Since there are no infections to suggest a primary source, I shall leave the choice between the first two for the present.

The relation of sulfonamides to liver disease has been under debate in the past. References in the literature tend to show that sulfonamides will damage the liver, particularly if there is liver damage already present, but in the majority of cases, there is little evidence of liver damage due to sulfonamides. I do not think the sulfonamides played any role in this case.

Whether or not there was a relation between the drop in fever and the penicillin therapy, it is difficult for me to say. I do not have any idea what the white-cell count was before admission. It is known that fever is often associated with biliary disease with obstruction and is not altered by penicillin.

The onset of jaundice is nearly conclusive evidence of increased obstruction to the extrahepatic system or of intrahepatic liver disease with possibly intrahepatic obstruction.

On physical examination the patient did not have the typical configuration of a cirrhotic person, which is that of a large abdomen and wasted chest, arms, legs and face. He was apparently well developed and well nourished. Certainly anyone who is so far advanced with alcoholic cirrhosis that he dies within a few days should manifest the physique of typical cirrhosis. Icterus is obvious from the van den Bergh reaction and from the history of jaundice. The second reason why I think he did not have cirrhosis is that there were no spider angiomias, and, by and large, the majority of cirrhotic patients show spider angiomas with liver damage, at least when they are as close to death as this man was. The tremendous enlargement of the liver is a third clue that this was not cirrhosis.

A patient with alcoholic cirrhosis dying of liver failure would have a normal-sized, or perhaps slightly enlarged, scarred liver. It is true that with an inadequate dietary intake there may be a massive, fatty infiltration of the liver, but such people are not usually so sick and the process can be reversed rather readily with proper diet. This man had a good diet, so that I do not think he had fatty infiltration of the liver.

What conditions could cause such a tremendous liver as this? One thinks immediately of cancer, primary or metastatic. It is generally accepted that primary carcinoma of the liver does not occur unless cirrhosis is present. However, if I recall correctly, I believe there was a case in this hospital of a primary hepatoma without evidence of cirrhosis. Is that correct, Dr. Mallory?

DR. TRACY B. MALLORY: There were 2 cases.

DR. CULVER: The absence of cirrhosis does not absolutely rule out a primary carcinoma of the liver cells, which can certainly cause a tremendous enlargement of the liver. This is one good possibility that we have to keep in mind in this case.

Metastatic cancer also may produce a tremendous liver. In this case we should suspect the gastrointestinal tract, although there were no symptoms to make it obvious, the pancreas or the biliary tract. I do not believe at the present time that we can differentiate between the last two. In this age group there seems to be a greater likelihood of carcinoma of the pancreas than of carcinoma of the biliary tree. I have no statistics to support that statement, but I have been aware of many patients in the late forties and early fifties with carcinomas of the pancreas.

One other diagnosis that occurs to me is a vascular tumor of the liver, a hemangioma; I recall a case of a tremendous liver on the basis of hemangioma. I should like to suggest that along with primary carcinoma and metastatic carcinoma as the three most likely possibilities to explain this man's liver disease.

The lack of irregularity of the liver is of some help in ruling out metastatic cancer but does not rule it out entirely. I do not believe it can rule out either one of the other two chief possibilities that I am considering, primary carcinoma of the liver or hemangioma. There was no palpable enlargement of the spleen, which is again evidence that there was probably not much fibrosis in the liver and probably not cirrhosis. There was no definite evidence of ascites, which is in favor of a tumor rather than primary intrahepatic liver disease.

A pyogenic involvement of the liver such as an abscess with an enlargement of this size — and I do not believe that the liver would have been this size — would produce a much higher fever, a higher white-cell count and much more toxicity than this man had when he entered the hospital.

The urinary findings, which are typical of regurgitation jaundice, do not help us much except to differentiate between hemolytic jaundice and all the rest of the jaundices. The hemoglobin suggests that the patient had not bled very much although he may have been dehydrated. The white-cell count was somewhat elevated, with an increase in neutrophils, which suggests an infection somewhere or might represent obstruction. I do not know what happens to the white cells in primary hepatoma or in hemangioma. The clay-colored stool may mean that there was an absence of bile pigment, or it might represent a pseudoacholic stool. The guaiac-positive stool may be explained by the prolonged prothrombin time, which is often used to explain bleeding in the gut, or it may have been secondary to a carcinoma of the head of the pancreas or in the region of the ampulla of Vater. The guaiac test on the stool in this case may be an important "tip-off" pointing to a primary lesion in the region of the ampulla — cancer of the ampulla or of the head of the pancreas.

The liver-function tests are those of extrahepatic liver disease. This is important, because it clinches the point so far as ruling out cirrhosis is concerned. We do not have a low albumin and a high globulin. We do not have a ++++ cephalin-flocculation test, but a ++ and a +++. A number of tests, on the other hand, very definitely favor extrahepatic obstruction — for example, a high alkaline phosphatase and a prolonged prothrombin time. If we knew the response to vitamin K, that would help us a little more. The nonprotein nitrogen, which we would expect to be low if there were intrahepatic liver disease, was normal. I do not believe a low normal serum amylase or a high normal lipase is of much diagnostic value in ruling in or ruling out carcinoma of the pancreas. There are numerous papers both pro and con, but in my experience these tests have not been helpful.

Are there any x-ray films? There were none reported in the protocol.

DR. STANLEY M. WYMAN: There are some, Dr. Culver.

The chest reveals no localized disease, but the heart shadow is somewhat prominent in the region of the left ventricle and the aorta is more tortuous than one would expect in a patient of this age. The film of the abdomen shows the liver shadow extending downward and overlying the iliac crest, lying far over in the left abdomen as described on physical examination. This is further confirmed by the barium enema, which shows no intrinsic disease, however. There is no evidence of enlargement of the spleen.

DR. CULVER: This man went downhill rapidly and died of what appears to have been liver failure, possibly from carcinoma.

To summarize this case, I do not believe in spite of the history that there was any evidence of portal or alcoholic cirrhosis. I believe that there was a primary hepatoma of the liver without cirrhosis, a metastatic cancer of the liver from the region of the head of the pancreas or ampulla, or a vascular tumor of the hemangiomatous type. How can we differentiate between these three? There was marked evidence of obstruction, and a positive guaiac test on the stool. I doubt very much if either a hemangioma or a primary hepatoma would produce that much obstruction, but they may. A tumor of the head of the pancreas would. I should like to make my diagnosis metastatic extension to the liver from carcinoma of the head of the pancreas or the ampulla of Vater; the second choice is a vascular tumor of the liver, of the hemangiomatous type.

A PHYSICIAN: I wonder if Dr. Culver would comment on the van den Bergh test.

DR. CULVER: My opinion about the van den Bergh test is that the direct value over the total value has little diagnostic use in differentiating intrahepatic from extrahepatic liver disease. I am sure that Ducci and Watson* would take exception to that statement. We have found no difference in the van den Bergh reaction in patients with cirrhosis or hepatoma on the one hand and those with obstructive jaundice on the other. I do not think it is of any diagnostic value whatsoever.

DR. ALFRED E. KRANES: Did all the service agree that this liver was smooth and regular?

DR. MALLORY: Yes; there was no argument about that.

DR. KRANES: That might be used as an argument against metastatic disease of the liver, although it is a notoriously inaccurate observation.

DR. MALLORY: Some of the people who had talked with the patient and with his friends were impressed with the grade of alcoholism saying that in a period of five and a half years he had never been out of the influence of alcohol at any moment.

DR. KRANES: The patient stated that he had a good appetite. Alcoholic patients always make that statement, but it is very difficult to check up on. They may think they are eating well.

DR. CULVER: You have seen, I am sure, and I have seen quite a number of alcoholic persons who have been heavy drinkers for years who we know have eaten well. I argue that this patient was that type because the physical examination suggests that he was a heavy drinker and a heavy eater.

DR. JACOB LERMAN: I wonder if one would not expect that bile stasis alone over a long period would cause this very large liver.

DR. CULVER: That is a very good point. I had not considered that perhaps as seriously as I should. We might use that as an argument in favor of something like either of my other two major diagnoses, a hemangioma or a primary hepatoma. I am not going to hedge at this point and agree on either of the latter possibilities.

*Ducci, H., and Watson, C. J. Quantitative determination of serum bilirubin with special reference to prompt-reacting and chloroform-soluble types. *J. Lab. & Clin. Med.* 30:293-300, 1945.

A PHYSICIAN: Could not the obstruction have been due to a metastasis at the porta hepatis from a hepatoma or from a carcinoma of the extrabiliary tract?

DR. CULVER: It could, but by and large I would not expect quite such a degree of obstruction unless there were a lesion right where the major hepatic ducts leave the liver.

CLINICAL DIAGNOSES

Cholemia.
Portal cirrhosis.
Metastatic liver disease.

DR. CULVER'S DIAGNOSIS

Carcinoma of head of pancreas (? ampulla of Vater), with metastases to liver.
? Vascular tumor of liver.

ANATOMICAL DIAGNOSIS

Carcinoma of head of pancreas, with metastases to regional lymph nodes and massive metastases to liver.

PATHOLOGICAL DISCUSSION

DR. MALLORY: Autopsy showed an enormous liver, weighing 6400 gm., which was almost completely replaced with carcinoma. This carcinoma was present in the form of multiple, often contiguous nodules replacing most of the liver parenchyma and in the form of tumor thrombi within many of the portal veins, the latter being a relatively uncommon finding with metastatic cancer whereas it is a very characteristic finding in hepatoma. However, in the small portions of the liver uninvolved by tumor there was no evidence of cirrhosis, and we did find an obvious primary tumor in the head of the pancreas so that I am quite confident that the tumor in the liver was entirely metastatic. There were a few lymph nodes in the region of the common bile duct that showed involvement, and many of the small pulmonary vessels contained microscopical emboli of tumor cells that had not become adherent to the vessel walls. There was no invasion of the pulmonary parenchyma, and I think these were merely a terminal event probably of only a few days' duration. There was a slight hypertrophy of the heart and a slight enlargement of the kidneys consistent with a bile nephrosis.

DR. KRANES: Was the surface of the liver smooth?

DR. MALLORY: No; it was moderately nodular with umbilicated nodules, but none of the projections were extremely large and the patient still had a fair amount of abdominal fat.

MASSACHUSETTS MEDICAL SOCIETY

PROCEEDINGS OF THE ONE HUNDRED AND SIXTY-NINTH ANNIVERSARY

MAY 16, 17 and 18, 1950

THE one hundred and sixty-ninth anniversary of the Massachusetts Medical Society was observed at the Hotel Statler in Boston on May 16, 17 and 18, 1950.

Seventeen hundred and forty-one physicians, 256 ladies, students, nurses and interns and 456 exhibitors were registered.

The Supervising Censors met in Parlor B at the Hotel Statler on May 15 at 4:30 p.m. This meeting was followed by the Cotting Supper in the Georgian Room, which was attended by 251 councilors. The annual meeting of the Council followed this supper at 7:00 p.m.

TUESDAY, MAY 16

The first general session opened at 9:00 a.m. in the Georgian Room of the Hotel Statler under the co-chairmanship of Drs. Daniel B. Reardon and John W. Spellman.

The one hundred and sixty-ninth annual meeting of the Society was held at 11:00 a.m. President Arthur W. Allen presided. The attendance was about 400. Dr. Allen spoke on "The State of the Society," after which the annual oration, "Safe Management of Fractures," was delivered by Dr. John W. O'Meara. (This lecture appeared in the May 18 issue of the *Journal*). The annual luncheon was served in Parlors A and B to 90 fellows.

The second general session was held at 2:00 p.m. under the co-chairmanship of Drs. Albert A. Hornor and John Homans.

WEDNESDAY, MAY 17

The third general session was held at 9:00 a.m. under the co-chairmanship of Drs. Peirce H. Leavitt and James M. Faulkner. The Shattuck Lecture was delivered at 11:05 a.m. by Dr. Merrill C. Sosman on the subject "The Specificity and Reliability of Roentgen Diagnosis." (This lecture appeared in the June 1 issue of the *Journal*.)

At noon, certain of the sections held their luncheons, followed by their annual meetings. The Section of Medicine, under the chairmanship of Dr. Allen S. Johnson, met in Parlor A with an attendance of 85.

The Section of Pediatrics met in Parlor B, with Dr. Stewart H. Clifford as chairman and about 58 in attendance. The Section of Radiology, under the chairmanship of Dr. Laurence L. Robbins, met in Parlor C, with 46 attending. The Section of

Physical Medicine, with Dr. Sidney Licht as chairman, met in the Hancock Room, with 22 in attendance. The Section of Ophthalmology and Otolaryngology met at the Hotel Bradford, and the Section of Industrial Health met on the fourth floor of the Hotel Statler, the former with Dr. James J. Regan as chairman and 78 in attendance, and the latter with Dr. Daniel L. Lynch as chairman and about 62 attending.

At 2:00 p.m. the fourth general session was held with Drs. Grantley W. Taylor and W. Fenn Hoyt as chairmen.

The annual dinner was held in the Georgian Room at 7:00 p.m. with 375 in attendance. The speaker of the evening was Claude M. Fuess, Ph.D., Litt.D. (hon.), who delivered an address entitled, "As Others See You." An award was presented to Mr. Thomas F. Costello, of Lowell, editor and publisher of the *Lowell Sun*, for outstanding work in the field of public relations.

Thursday, May 18

The fifth general session was held at 9:00 a.m. under the joint chairmanship of Drs. Curtis C. Tripp and Charles G. Shedd. At noon the remaining scientific sections held their luncheons and meetings. The Section of Surgery met in the Salle Moderne, with Dr. Franklin G. Balch, Jr., as chairman, and about 31 in attendance. The Section of Obstetrics and Gynecology, under the chairmanship of Dr. Daniel McSweeney, met in Parlor A, with 38 attending. The Section of Dermatology and Syphilology met in Parlor C, with Dr. Maurice M. Tolman as chairman and an attendance of 34. The Section of Anesthesiology met in Parlor B, with Dr. Jacob Fine as chairman and 40 attending. The Section of Physiology and Pathology and the Section of Psychiatry and Neurology both met on the fourth floor, the former with Dr. Donald A. Nickerson as chairman and about 30 attending, and the latter with Dr. Walter E. Barton as chairman and an attendance of 83. The sixth general session was held at 2:00 p.m. under the co-chairmanship of Dr. W. Richard Ohler and Dr. Allen S. Johnson.

An exhibition of works of art by members of the Massachusetts Physicians' Art Association was on view throughout the meeting.

The list of officers, standing and special committees, councilors, censors, admissions and deaths is appended.

H. Quimby Gallupe, *Secretary*

Annual Meeting of the Society

The one hundred and sixty-ninth annual meeting of the Massachusetts Medical Society was called to order by the president, Dr. Arthur W. Allen, in the Georgian Room of the Hotel Statler, Boston, at 11:00 a.m. on May 16, 1950.

The Secretary submitted the record of the annual meeting held on May 24, 1949, in Worcester as published in the July 28, 1949, issue of the *New England Journal of Medicine*, and moved its acceptance.

The motion was seconded, and it was so voted.

The President then called upon Dr. Edward P. Bagg, chairman of the Committee on Council Rules and By-laws, who addressed the meeting as follows:

> Since ultimate authority in the matter of by-laws lies in the Society itself, certain changes are submitted at this meeting for action. These changes have been framed by the Committee on Council Rules and By-laws, edited and approved by previous votes of the Council.
>
> In order that every district should be enabled to meet the requirements of the by-laws as to representation on standing committees, a new sentence was added to the third paragraph of Section 5, Chapter 3 of the By-laws — namely, "each district shall elect no fewer than six councilors."
>
> At the request of the Committee on Membership, Section 2 (a) of Chapter 5 was amended by the insertion, after "residence" in the first sentence, of the words "or place of practice." Thus, frequent transfers from one district to another will be avoided. Option is left with the applicant. The phrase now will read, "an applicant whose legal residence or place of practice is within the district of that Society."
>
> Since the influx of displaced physicians has abated, and also because one year's practice among his fellows is more revealing than five would be elsewhere, Section 2 (b) of Chapter V, concerning the requirement of length of residence, was changed from "United States or its territories for at least five years" to "district from which he makes application for at least one year."
>
> The request also was approved that a member of the Society who is a president or past president of the American Medical Association be admitted *ipso facto* to membership in the Council. The Committee is aware that further addition of members to the Council should be limited because of an already large membership, but embarrassment from this source is not probable and is offset by freedom from that of omitting honor where honor is due.

Dr. Bagg then made a motion that Chapter V, Section 2 (a) be amended by the insertion of the words "or place of practice" after "residence" in the first sentence. The motion was seconded.

Dr. Barton (Norfolk) said the councilors of Norfolk had voted to disapprove this change in the by-laws because legal residence is more permanent.

Dr. Henry F. Howe (Norfolk South) argued that fellows should attend meetings of the districts where their offices are maintained.

Dr. Carl Bearse (Norfolk) presented, among other arguments against the change, one that doctors' wives should join the auxiliary that included their home town.

Dr. Leland S. McKittrick (Suffolk) favored the change because it gave the doctor a choice of district society — a choice that he should have.

The President called for a vote, and the motion was carried.

Dr. Bagg (Hampden) then made a motion that Chapter V, Section 2 (b), be amended by the substitution of the words "district from which he makes application for at least one year" for the words in the second paragraph "United States or its territories for at least five years."

The motion was seconded, and it was so voted.

Dr. Bagg then made a motion to amend the first paragraph of Chapter IV, Section 1, after the

comma following the word "medicine," by deletion of the remainder of the sentence and substitution of the following: "as well as the president and past presidents of the American Medical Association, the president of the Massachusetts Medical Service and the director of the Massachusetts Medical Service, provided they are members of this society."

The motion was seconded, and it was so voted.

He then moved approval of the report as a whole. The motion was seconded, and it was so voted.

The Secretary reported on the membership of the Society as follows:

The membership of the Massachusetts Medical Society in May, 1949, was 6752. This has been increased by 302 new members admitted since then. In addition, there were 5 reinstatements, bringing the total to 7059.

Losses were as follows: 35 by deprivation, 40 by resignation and 120 by death, which brings the sum total as of May 16, 1950, to 6864 members.

Dr. Allen then asked Dr. Albert Hornor, the vice-president, to assume the chair.

Dr. Hornor (Suffolk) presented Dr. Allen, who gave the following address on the state of the Society:

During the past twelve months the Society has been busy dealing with many important problems that affect us individually and collectively. The permanent staff, officers and committeemen have worked diligently and effectively in your behalf. We believe that progress has been made and that the Society's welfare has been safeguarded to the best of our various abilities. Major issues have been met with foresight and expediency. It would not be possible to give you all the details in this report, and some of the items of interest may be omitted unwittingly. I shall try, however, to review the year's work from the standpoint of importance as I see it. Those of you who think that greater stress should have been made on any particular phase of the Society can obtain full records from the Secretary. I shall not strictly adhere to any schedule of priority or necessarily follow a chronologic order.

At this one hundredth and sixty-ninth annual session we have the initial appearance of four new sections — namely, Physiology and Pathology, Psychiatry and Neurology, Industrial Health and Ophthalmology and Otolaryngology. The pattern of our parent organization has been followed in the naming of these additional sections, and it appears that these will be well attended and will increase the interest of the fellows in the program.

One of the accomplishments of the Society this year was the adoption of the thirteen principles for which we stand. These were based largely on principles already approved by us in 1946. Rewording and rephrasing, with additions and deletions, required careful thought and considerable time of a large and interested group of fellows. Most of these principles were accepted by the Council at its annual meeting in May, 1949, but the final product was not adopted until the October meeting. With these standards before us it has been possible to deal with legislative problems on a sound basis of policy.

The committees on National Legislation and Economics have met jointly at frequent intervals to discuss the various ways and means by which we can improve patient care. There has been a tolerant attitude regarding the issues at stake, and many experts from the Society have been called in by the committees for advice.

It has been evident that direct contact with our congressman was helpful in defeating the Administration's Plan I. Also, we believe that our reasons for advocating changes in much of the proposed legislation were seriously considered by our lawmakers. These men have been open-minded and responsive. We hope that the work of our own members in this field will result in improvement in the health of our people and that this can be accomplished by physicians. We have stressed voluntary plans and opposed compulsory measures. We realize that there are limitations that must be endured and that, for the best interests of the people, progress must be gradual and not precipitous. We must try for changes that are within the scope of our personnel. It is our duty to endeavor to bring about the best medical care that we are capable of producing. Most of us do not believe that this can be best accomplished by federal control of medicine in the United States. All physicians are willing and anxious to aid the Government in every conceivable way to bring about the sanest and most feasible plans to increase facilities for the continued improvement of our already excellent status of health and medical service.

The Committee on Legislation has had a successful year. No measures that we have opposed have so far passed. To the committeemen, to our legal adviser, to the Director of Medical Information and Education and to many of our fellows we express our thanks.

The Auxiliary has also been helpful and interested in these affairs. It is believed that this new adjunct to the Society will always stand ready to improve our efforts in all fields of education, legislation and better health measures as well as patient care.

The Pilot Clinic authorized by you at the last annual meeting has functioned well. The amazing demand for this complete type of examination indicates that there are many people in this state who are health conscious. Our project has taken fewer people for, as nearly as possible, complete screening than many of the clinics operating elsewhere. From this rather thorough work we will have made a contribution to research in health that will be of great value. Practical deductions can be drawn that will aid our successors in determining the needs of the people relative to preventive medicine. We are grateful to the New England Center Hospital for the use of their facilities in carrying out this study.

Confusion has always existed about so-called publicity regarding the activities of physicians. The blanket disapproval of advertising by physicians has at times made it difficult to decide whether or not there has been a break in the code of ethics. Censure for any publicity was possible regardless of how it came about, causing many physicians to withhold news particularly if it involved their names. This resulted in considerable dissatisfaction on the part of newspapermen and certainly played a role in what has been termed an "unfriendly press." Since there were cases in which the news value concerned a physician's duties, more especially when these applied to outstanding progress in the treatment of certain disorders, an ethical method of releasing this news was needed. A special committee developed the publicity code, and this was adopted by you recently. This has been incorporated in the new edition of the rules and by-laws.

Other changes in the by-laws consist of two important features: since 6 appointments from each district on various standing committees are required, it seemed wise to have a mandatory minimum of 6 councilors appointed from each district society (this recommendation was adopted and has been put into effect); previously we had ruled that graduates of foreign and unapproved medical schools must wait five years before making application for membership in the Society. This waiting period had no relation to residence in Massachusetts. Furthermore, the sources of graduates from unapproved American medical schools have been closed. The Committee on Membership therefore believed that a waiting period of one year with residence in this state would better meet the problem. This recommendation was adopted and is now in force. It should not be forgotten that each applicant for membership is carefully screened by the district committee before consideration is given by the Committee on Membership.

The new *Directory* has many improvements and we are indebted to the Secretary for bringing about these changes. It contains now a list of the towns and cities of the State, indicating the district to which they belong. Each fellow's address is given, and by a code system his

specialty is tabulated. The fact that these data are always at hand has enhanced the efforts of our headquarters group in dispensing much-sought-for information.

We have had a committee to meet with the Massachusetts Hospital Association. Since there is a definite need for closer co-operation between physicians and hospitals this committee's name has been changed to that on Hospital and Professional Relations. Meetings of this committee with a committee from the Massachusetts Hospital Association have resulted in a better understanding of our mutual problems. Preceded by a questionnaire, an effort will be made to bring the staff, the administration and the trustees of hospitals into closer relation. It is apparent by so doing that the patient's best interest can be safeguarded.

The Public Health Conference, held June 15, 16 and 17, 1949, and the Industrial Health Conference, held December 14, 1949, were outstanding features of specific activity during the year. Remarkable interest was evident, and the attendance was large at both conferences. Commendation for excellence is in order for the members of our committees in these fields for bringing about these useful meetings.

Graduate education has continued to make its important contribution to the welfare of the Commonwealth. Programs have been carefully planned and carried out by the Committee on Post-graduate Medical Education. Attendance has been large, and unusual interest demonstrated. It is not possible to estimate the hidden value of this type of effort on the part of the Society. The continued willingness to "teach gladly" is one point of evidence for the maintenance of a free society that cannot be discounted.

There has been much fanfare concerning the establishment of so-called "grievance committees." This has been heralded as a new idea, and doubtless it may be new in some areas. The Massachusetts Medical Society's Committee on Ethics and Discipline was established in 1871 and has functioned effectively in this state as a grievance committee. It was apparent that there was no need in Massachusetts for new committees to deal with such problems. It was believed, however, that perhaps the public and the members of the profession could be made more aware of the functions of this committee. The Director of Medical Information and Education has been empowered to correct any fault that may exist in this regard.

The Massachusetts Medical Service, Inc. (Blue Shield), established a decade ago, has had a very successful record. Careful planning and sound principles have resulted in satisfaction to the subscriber and to the physician. Since the limits of income for complete service contract had not kept pace with increasing wages a second change in policy was indicated. After careful consideration of the whole problem the Council, at its February session, voted to approve a new contract. In addition to the present $3000 limit, with unchanged premiums and disbursements, it was agreed to insure the group with incomes between $3000 and $5000, the new group to pay an increased premium with the professional service payments increased proportionally. Originally, Blue Shield had a family limitation of $2500 per year. At that time such a contract covered approximately 80 per cent of the population. As income increased it was necessary to elevate these limits to the $3000 level in order to continue to cover the same percentage of the population. This change was made with little or no increase in professional fees. The new contract will cover more than 80 per cent but less than 90 per cent of the population. Therefore, the purpose of the original Blue Shield concept is maintained. There is a general opinion that this new group to be covered is a justifiable undertaking. It is expected that all details will have been sufficiently settled by July 1 of this year to allow this new venture to go on trial.

The Committee on Public Relations, after much discussion, brought before the Council a recommendation that significant cognizance be taken of lay people who had made outstanding contributions to the welfare of humanity. It was voted to adopt this recommendation and to authorize "The Massachusetts Medical Society Lay Award." This new concept has been carried into effect, and Mr. Thomas F. Costello, editor of the *Lowell Sun*, has been selected as the first recipient of this honor. Through his efforts editorially and otherwise we believe that he has more than fulfilled the requisites for this recognition. The Society should be justly proud of this selection specifically and duly impressed by the soundness of the policy as a whole.

The Committee on Society Headquarters, with paid expert advice, has thoroughly investigated all possible locations for a permanent Society home. After full consideration of all factors the property at 22 Fenway appears to be the logical choice. This house adjoins the Boston Medical Library and can be connected with that building on at least two and possibly three levels. It is large enough for our immediate needs, and there is a vacant lot in the rear of the building that allows for future expansion. One only needs a glance at our present overcrowded space to realize how urgent this new building has become. We can no longer expect our permanent staff, our officers and our committees to give us efficient service with radical changes. Fortunately, we have enough in our building fund to cover the cost of the purchase and partially to take care of the expense of alterations. With an additional $50,000 from our reserve fund allotted to this project we will have a headquarters that not only will be adequate but of which we can be justly proud.

The Boston Medical Library is the keystone of the Massachusetts Medical Society. Without it our fellows and the students of medicine in our vicinity would be greatly handicapped. More and more use of the Library by the members of the Society during the past few years has justified our earlier vote for support. Now that we have made our headquarters in an adjoining building it is believed that our interest will be increased. This makes it possible for the Library trustees to obtain new funds for renovation and for completion of the stacks long needed. John Ware Hall can be made a more suitable room for our meetings. It will be possible to join the third floor of our building with this auditorium at some not too distant future date. This project should be made a matter of our concern.

The financial condition of the Society is sound. We have lived within our means, but this has made it necessary for the Committee on Finance to reduce the budgets in many departments for the present year. Few of the fellows realize how their $25 annual dues are allotted: $5 goes to the Library, $1.50 to the Benevolent Fund, and $3 to the *Journal* (representing approximately half the cost to publish your subscription), and the *Journal* further does approximately $5000 a year of our printing, which we get free. If this amount were added to the *Journal's* budget we should allot it actual cost of each subscription as its just due. Since the Society owns the *Journal* it matters little how its costs are covered. Few, if any, organizations that publish a scientific journal give free subscriptions or any reduction in subscriptions to their members. This is the chief reason many journals are reported to be making money while we operate on a narrow margin. We have in the past returned about $8000 to the districts, making a further reduction in our dues, so that about $14 per fellow is left for running expenses.

We have a reserve fund of about $250,000. Do the fellows wish to dig into this and begin spending it now for current needs? We are in need of more funds for many of our projects. Our department of information and education needs expansion particularly. We cannot take on any new worthy causes with our present dues. Many states have dues much higher than our own. Few require less, and the average of all the states is $32. Few states have a journal of more than local fame, and none has one comparable to ours. My belief is that you must consider these problems seriously. Should not our dues be $35 next year?

In closing I wish to say that I have been honored to serve you. The year of apprenticeship as president-elect was largely educational. This year of presidency has been a fruitful experience. I take this opportunity to thank you all for your help, your consideration and your response to my calls for guidance. I wish you to know that I believe that few state societies, if any, have as reliable a permanent staff, as conscientious committees and as thoughtful co-operative fellows as this of ours. May the

years to come bring us greater renown in our effort to serve humanity!

Dr. Hornor (Suffolk) then spoke as follows:

I should like very much to express the thoughts that I know are in the minds of every one of you: that this society has in the past year made great progress and we are indebted to Dr. Allen for the amount of effort, thought and sympathy that he has put forth while he has worked on this task. He has been busy at it many hours of the day and night, and on many nights of every week. I think the Society owes Dr. Allen a great vote of thanks.

The President resumed the chair and requested the Secretary to read the list of fellows who had been active members for fifty years or more. The Secretary read the following list of names:

Charles F. Atwood
George G. Averill
John W. Bartol
Michael F. Burke
Henry D. Chadwick
Rockwell A. Coffin
George P. Cogswell
Anna W. Croacher
Augustus W. Dudley
George W. Ewing
Joel E. Goldthwait
George H. Gray
Nelson C. Haskell
Thomas H. Heaton
Elliott P. Joslin
Walter B. Lancaster
George A. Leahey
Arthur C. Lewis
Felix F. McGirr
Charles E. Mongan
Hardy Phippen
Boyden H. Pillsbury
William H. Robey
Arthur J. Shaw
Charlotte S. Smith
Marsena P. Smithwick
Sarah R. Stowell
Franklin W. White
Michael W. White
George C. Wilkins
Edward R. Williams
Alfred Worcester

Those who were able to be present came to the platform to be presented with a gold pin and to be congratulated by Dr. Allen.

The President then called upon the delegates from the other New England state medical societies. Dr. Richards responded for New Hampshire, Dr. Hall for Vermont, and Drs. Smith and Burlingame for Connecticut.

Dr. Allen thanked the delegates for their messages of good will.

He then introduced Dr. Chester S. Keefer (Suffolk), who spoke as follows:

I have the honor to present three men for the Massachusetts Medical Society student prizes and certificates of merit. These prizes and certificates of merit have been made possible through the generous contribution of one of our members who requests that his name remain anonymous.

We have been instructed that these certificates and prizes are to be given to a graduating student from each of the three Boston medical schools — Boston University School of Medicine, Harvard Medical School and Tufts College Medical School — for "Diligent work as a student and for qualities which serve to designate him as a good physician."

This year the prize consists of three books: two volumes, first published in 1919 as a limited edition, of contributions to medicine and biology, to commemorate the seventieth birthday of Sir William Osler; and a third, small book commemorating the one hundredth anniversary of his birth. In selecting this prize the Committee wanted to show by example some of the qualities of a good and great physician of the last century.

In turning these prizes and certificates over to the President for presentation I want to read a little verse that was written by the Indian dramatist Caladossa. This verse, which was found among the papers in Sir William Osler's desk after his death, is entitled "Salutation to the Dawn":

"Look to this Day!
For it is life, the very Breath of Life.
In its brief course lie all the Realities of your Existence;
The Bliss of Growth,
The Glory of Action,
The Splendor of Beauty;
For Yesterday is already a Dream, and Tomorrow is only a Vision;
But Today well lived makes every
Yesterday a Dream of Happiness, and every
Tomorrow a Vision of Hope.
Look well therefore to this day!"

Dr. Keefer then introduced the three recipients, Joseph Franklin, Leonard Cronkhite, Jr., and Miles Lee Perkins, Jr. Dr. Allen presented the awards, congratulating the three students and reading the citation:

This is a Certificate of Merit, Massachusetts Medical Society, on the occasion of the 169th Annual Meeting of the Massachusetts Medical Society. Be it known that these students of medicine have been selected to receive this Certificate of Merit for diligent work as students and for qualities which serve to designate them as good physicians.

The President then introduced the newly elected officers of the Society: president, Dr. Leland S. McKittrick, president-elect, Dr. W. Richard Ohler, vice-president, Dr. Patrick J. Sullivan, secretary, Dr. H. Quimby Gallupe, treasurer, Dr. Eliot Hubbard, Jr., and assistant treasurer, Dr. Norman A. Welch.

Dr. Allen then introduced Dr. John W. O'Meara of Worcester, who delivered the one hundred and forty-second annual oration, entitled "Safe Management of Fractures."

At the close of the oration Dr. Allen declared the one hundred and sixty-ninth annual meeting adjourned at 12:55 p.m.

Officers for 1950-1951

President: Leland S. McKittrick, Brookline, 1180 Beacon St.
President-Elect: W. Richard Ohler, Jamaica Plain. Office, Boston, 319 Longwood Ave.
Vice-President: Patrick J. Sullivan, Dalton, 471 Main St.
Secretary: H. Quimby Gallupe, Waltham. Office, Boston, 8 Fenway.
Treasurer: Eliot Hubbard, Jr., Cambridge, 29 Highland St.
Assistant Treasurer: Norman A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Ave.
Orator: Donald Munro, Milton. Office, Boston, 818 Harrison Ave.

Committees Elected by the Districts

Executive Committee of the Council — Established 1941 (members *ex-officiis* and one councilor and alternate elected by the councilors of each district medical society).

President: Leland S. McKittrick, Brookline, 1180 Beacon St.
President-Elect: W. Richard Ohler, Jamaica Plain. Office, Boston, 319 Longwood Ave.
Vice-President: Patrick J. Sullivan, Dalton, 471 Main St.
Secretary: H. Quimby Gallupe, Waltham. Office, Boston, 8 Fenway.
Treasurer: Eliot Hubbard, Jr., Cambridge, 29 Highland St.
Assistant Treasurer: Norman A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Ave.

Term Expires 1951

Essex South: Albert E. Parkhurst, Beverly, 1 Monument Square. (Alternate: Edwin D. Reynolds, Danvers, 48 High St.)
Hampshire: Maurice T. Kennedy, Hadley, 11 Middle St. (Alternate: L. Beverley Pond, Easthampton, 115 Main St.)
Middlesex South: Joseph C. Merriam, Framingham, 198 Union Ave. (Alternate: John F. Casey, Brighton. Office, Boston, 475 Commonwealth Avenue.)
Norfolk South: Robert L. Cook, Quincy, 1245 Hancock St. (Alternate: William R. Helfrich, Quincy, 272 Southern Artery.)
Suffolk: Harvey A. Kelly, Winthrop, 200 Pleasant St. (Alternate: William E. Browne, Boston, 587 Beacon St.)
Worcester: John Fallon, Worcester, 10 Institute Road. (Alternate: Nicholas S. Scarcello, Worcester, 1 Selden St.)

Term Expires 1952

Barnstable: Harold F. Rowley, Harwich Port. (Alternate: Donald E. Higgins, Cotuit, Main St.)
Bristol North: William M. Stobbs, Attleboro, 63 Bank St. (Alternate: Milton E. Johnson, Attleboro, 33 Bank St.)
Bristol South: Curtis C. Tripp, New Bedford, 416 County St. (Alternate: John C. Corrigan, Fall River, 422 North Main St.)
Essex North: Arnold P. George, Haverhill, 31 Summer St. (Alternate: Frederic N. Sweetsir, Merrimac, 18 Main St.)
Middlesex East: Thomas P. Devlin, Stoneham, 33 Pleasant St. (Alternate: Robert Dutton, Wakefield, 33 Avon St.)
Plymouth: Alton L. Hurlburt, East Bridgewater, 81 Central St. (Alternate: James M. Bonnar, Middleboro, 33 Pearl St.)

Term Expires 1953

Berkshire: Helen M. Scoville, Pittsfield, Pittsfield General Hospital. (Alternate: J. Harry Fierman, Pittsfield, 74 North St.)
Franklin: John Moran, Greenfield, 15 Franklin St. (Alternate: Rosario Charron, Greenfield, 308 Main St.)
Hampden: Frederic Hagler, Springfield, 20 Maple St. (Alternate: George B. Corcoran, West Springfield, 84 Park St.)
Middlesex North: Artemas Stewart, Bowell, 310 Merrimack St. (Alternate: Leonard Hall, Lowell, 8 Merrimack St.)
Norfolk: Basil E. Barton, West Roxbury, 10 Richwood St. (Alternate: Timothy F. P. Lyons, Boston, 270 Commonwealth Ave.)
Worcester North: James V. McHugh, Leominster, 55 West St. (Alternate: Kurt K. Jolma, Gardner, 86 Jonathan St.)

Subcommittee on Blue Cross-Blue Shield Problems

Curtis C. Tripp, Bristol South, *chairman*; Basil E. Barton, Norfolk; Harvey Kelly, Suffolk; John Fallon, Worcester; Joseph C. Merriam, Middlesex South.

Committee on Nominations — Established 1874 (one councilor and alternate elected yearly by each district medical society).

Barnstable: Donald E. Higgins, Cotuit, Main St. (Alternate: Harold F. Rowley, Harwich Port.)
Berkshire: Helen M. Scoville, Pittsfield, Pittsfield General Hospital. (Alternate: J. Harry Fierman, Pittsfield, 74 North St.)
Bristol North: Joseph L. Murphy, Taunton, 23 Cedar St. (Alternate: Curtis B. Kingsbury, Taunton, 63 Prospect St.)
Bristol South: Harold E. Perry, New Bedford, 159 Cottage St. (Alternate: William Mason, Fall River, 151 Rock St.)
Essex North: Charles F. Warren, Amesbury, 155 Main St. (Alternate: Percy J. Look, Andover, 115 Main St.)
Essex South: C. F. Twomey, Lynn, 80 Ocean St. (Alternate: DeWitt Clark, Salem, 2 Oliver St.)
Franklin: Warren Thomas, Montague, Central St. (Alternate: Wilton Deane, East Northfield, 185 Main St.)
Hampden: George L. Steele, Springfield, 20 Maple St. (Alternate: Edward P. Bagg, Holyoke, 207 Elm St.)
Hampshire: Joseph R. Hobbs, Williamsburg, Main St. (Alternate: L. Beverly Pond, Easthampton, 115 Main St.)
Middlesex East: John M. Wilcox, Woburn, 6 Bennet St. (Alternate: Harry L. Mueller, Winchester, 31 Church St.)
Middlesex North: Harry R. Coburn, Lowell, 202 Merrimack St. (Alternate: William Collins, Lowell, 174 Central St.)
Middlesex South: Fred R. Jouett, Cambridge, 1 Craigie St. (Alternate: Henry W. Godfrey, Auburndale, 14 Hancock St.)
Norfolk: Carl Bearse, Boston, 483 Beacon St. (Alternate: Herbert A. Rice, Canton, 472 Washington St.)
Norfolk South: Robert L. Cook, Quincy, 1245 Hancock St. (Alternate: William R. Helfrich, Quincy, 272 Southern Artery.)
Plymouth: Peirce H. Leavitt, Brockton, 129 W. Elm St. (Alternate: Donald A. Martin, South Hanson, Plymouth County Hospital.)
Suffolk: Conrad Wesselhoeft, Boston, 315 Marlboro St. (Alternate: Charles C. Lund, Boston, 20 Gloucester St.)
Worcester: F. Benjamin Carr, Worcester, 27 Elm St. (Alternate: George W. Ballantyne, Worcester, 27 Elm St.)
Worcester North: James V. McHugh, Leominster, 55 West St. (Alternate: James G. Simmons, Fitchburg, 30 Myrtle Ave.)

Committee on Public Relations — Established 1939 (one councilor elected yearly by each district medical society; the president and president-elect of the Society are chairman and vice-chairman, respectively; and the vice-president and secretary of the Society are members *ex-officiis*.)

Barnstable: Henry F. Hopkins, Chatham, Box 747.
Berkshire: Patrick J. Sullivan, Dalton, 471 Main St.
Bristol North: Milton E. Johnson, Attleboro, 33 Bank St.
Bristol South: Milton T. MacDonald, New Bedford, 99 Clinton St.
Essex North: Harold R. Kurth, Lawrence, 57 Jackson St.
Essex South: Bernard Appel, Lynn, 281 Ocean St.
Franklin: Lawrence R. Dame, Greenfield, 78 Federal St.
Hampden: Arthur H. Riordan, Springfield, 20 Maple St.
Hampshire: Joseph R. Hobbs, Williamsburg, Main St.
Middlesex East: George A. Marks, Winchester, 15 Dix St.
Middlesex North: Samuel Dibbins, Lowell, 528 Andover St.
Middlesex South: Ralph Wells, Lexington, 1430 Massachusetts Ave.
Norfolk: Dean S. Luce, Canton, 553 Washington St.
Norfolk South: Daniel B. Reardon, Quincy, 1245 Hancock St.
Plymouth: John C. Angley, Bryantville, School St.
Suffolk: G. Lynde Gately, East Boston, 624 Bennington St.
Worcester: Nicholas Scarcello, Worcester, 1 Selden St.
Worcester North: Donald B. Cheetham, Athol, 164 Exchange St.

Subcommittees of Committee on Public Relations

Tax-Supported Medical Care — Established 1940.
Albert A. Hornor, Suffolk, *chairman*; William W. Babson, Essex South; Donald Hight, Worcester; Francis P. McCarthy, Norfolk.

Committee to Meet with the Medical Advisory Committee of the Industrial Accident Board — Established 1942.
Gordon Morrison, Middlesex South, *chairman*; Charles H. Bradford, Suffolk; Joseph H. Burnett, Middlesex South; Somers Fraser, Suffolk; Laurence A. Putnam, Hampden.

Committee on Legislation — Established 1942 (one councilor elected yearly by each district medical society.

BARNSTABLE: A. J. D'Elia, Harwich Port, Main St.
BERKSHIRE: Thomas D. Jones, Pittsfield, 18 Bank Row.
BRISTOL NORTH: Wm. M. Stobbs, Attleboro, 63 Bank St.
BRISTOL SOUTH: Daniel F. Gallery, Fall River, 151 Rock St.
ESSEX NORTH: Lucien R. Chaput, Haverhill, 3 Washington Sq.
ESSEX SOUTH: Paul R. Hinchey, Salem, 355 Essex St.
FRANKLIN: Harold Mahar, Orange, 1 High St.
HAMPDEN: Alfred M. Glickman, Springfield, 95 State St.
HAMPSHIRE: John J. Curran, Northampton, 16 Centre St.
MIDDLESEX EAST: Charles R. Baisley, Reading, 46 Woburn St.
MIDDLESEX NORTH: Leonard Hall, Lowell, 8 Merrimack St.
MIDDLESEX SOUTH: Alice M. Broadhurst, Watertown, 259 Mt. Auburn St.
NORFOLK: Paul J. Jakmauh, South Boston, 300 Broadway.
NORFOLK SOUTH: David L. Belding, Hingham, 215 Main St.
PLYMOUTH: Harold H. Hamilton, Plymouth, 70 Court St.
SUFFOLK: William E. Browne, Boston, 587 Beacon St.
WORCESTER: John B. Butts, Worcester, 24 Franklin St.
WORCESTER NORTH: Joseph P. Marnane, Gardner, 4 Comee St.

Subcommittee of the Committee on Legislation

NATIONAL LEGISLATION — Established 1946.

Charles G. Hayden, Norfolk; *chairman*; Elmer S. Bagnall, Essex North; James G. Simmons, Worcester North; David L. Bending, Norfolk South; Vlado A. Getting, Middlesex South; Harold M. Frost, Suffolk; William H. Sweet, Suffolk.

STANDING COMMITTEES FOR 1950–1951

ELECTED BY THE COUNCIL MAY 15, 1950

Publications — Established 1825.

Richard M. Smith, Suffolk	June 6, 1933 (appointed chairman May 21, 1941)
Oliver Cope, Middlesex South	May 21, 1941
John Fallon, Worcester	November 14, 1944
James P. O'Hare, Suffolk	June 9, 1936
Conrad Wesselhoeft, Suffolk	June 2, 1937

Arrangements — Established 1849.

Gordon A. Donaldson, Middlesex South	May 19, 1947 (appointed chairman May 15, 1950)
John W. Norcross, Middlesex South	May 19, 1947
Albert Ehrenfried, Norfolk	May 24, 1948
Alexander Marble, Suffolk	May 15, 1950
Richard Warren, Suffolk	May 15, 1950

Ethics and Discipline — Established 1871.

Ralph R. Stratton, Middlesex East	June 9, 1936 (appointed chairman May 21, 1941)
William J. Brickley, Suffolk	February 3, 1947
Guy Richardson, Essex North	May 15, 1950
Allen G. Rice, Hampden	June 1, 1938
James H. Townsend, Middlesex South	May 23, 1949

Medical Education — Established 1881.

James M. Faulkner, Norfolk	May 21, 1946 (appointed chairman May 15, 1950)
C. Stuart Welch, Norfolk	May 15, 1950
Oliver Cope, Middlesex South	May 23, 1949
Isaac R. Jankelson, Norfolk	May 25, 1942
Robert T. Monroe, Norfolk	May 23, 1949

Membership — Established 1897.

Lewis S. Pilcher, Middlesex South	July 26, 1946 (appointed chairman May 24, 1948)
William A. R. Chapin, Hampden	May 23, 1945
Henry F. Howe, Norfolk South	May 23, 1949
Donald Munro, Suffolk	May 23, 1949
Samuel N. Vose, Suffolk	March 15, 1944

Public Health — Established 1912.

Roy J. Ward, Worcester	May 22, 1944 (chairman)
Alfred E. Frechette, Norfolk	May 23, 1949
Hugh Leavell, Middlesex South	May 15, 1950
Warren R. Sisson, Suffolk	May 19, 1947
Conrad Wesselhoeft, Suffolk	July 27, 1944

Subcommittees of the Committee on Public Health

MENTAL HEALTH — Established 1947.

Walter E. Barton, Norfolk	February 5, 1947 (chairman)
G. Colket Caner, Suffolk	May 23, 1949
William Malamud, Worcester	February 5, 1947
Francis M. Rackemann, Suffolk	May 23, 1949
Henry A. Tadgell, Hampshire	February 5, 1947

CHILD HEALTH — Established 1949.

James M. Baty, Middlesex South	October 1, 1949 (chairman)
Merritt B. Low, Franklin	October 1, 1949
Ralph A. Ross, Middlesex South	October 1, 1949
Lendon Snedeker, Suffolk	October 1, 1949
George Starbuck, Bristol South	October 1, 1949

Arthritis and Rheumatism — Established 1950.

Theodore B. Bayles, Suffolk	February 1, 1950 (chairman)
Charles E. Ayers, Worcester	February 1, 1950
Laurence D. Chapin Hampden	February 1, 1950
Franklin K. Paddock, Berkshire	February 1, 1950
Charles L. Short, Suffolk	February 1, 1950
Henry D. Stebbins, Essex South	February 1, 1950

Medical Defense — Established 1927.

Horatio Rogers, Suffolk	June 7, 1939 (appointed chairman May 19, 1947)
Charles B. Burbank, Norfolk	May 23, 1949
John E. Moran, Franklin	May 19, 1947
Charles J. Kickham, Norfolk	May 21, 1946
Philip Le Compte, Norfolk	May 15, 1950

Finance — Established 1938.

Robert W. Buck Middlesex South	May 21, 1946 (chairman)
Henry H. Faxon, Norfolk	May 23, 1949
Francis C. Hall, Suffolk	July 8, 1943
Fabyan Packard, Middlesex South	May 21, 1946
Bancroft C. Wheeler, Worcester	May 21, 1946

Society Headquarters — Established 1942.

Frank R. Ober, Suffolk	May 22, 1944 (appointed chairman Nov. 1, 1944)
Joseph S. Barr, Middlesex South	May 23, 1949
Albert A. Hornor, Suffolk	November 6, 1944
Dwight O'Hara, Middlesex South	May 23, 1949
Walter G. Phippen, Essex South	May 21, 1946

Industrial Health — Established 1942.

Henry C. Marble, Suffolk	May 19, 1947 (appointed chairman May 23, 1949)
Karl T. Benedict, Worcester	May 23, 1949
Harriet L. Hardy, Middlesex South	May 23, 1949
Daniel L. Lynch, Norfolk	May 25, 1942
George E. Morris, Norfolk	May 23, 1949

Albert O. Seeler	May 15, 1950
Middlesex South	
George F. Wilkins, Norfolk	May 23, 1949

Advisory Committee to Committee on Industrial Health — Established 1942.

Philip Drinker
William H. Seymour
Emma S. Tousant

Benevolence — Established 1948.

Dwight O'Hara,	May 24, 1948 (chairman)
Middlesex Sooth	
Theodore L. Badget, Norfolk	May 24, 1948
Robert W. Buck,	May 24, 1948
Middlesex South	
Eliot Hubbard, Jr.,	May 24, 1948
Middlesex South	
Charles C. Lund, Suffolk	May 24, 1948

Council Rules and By-Laws — Established 1948.

Edward P. Bagg, Hampden	October 6, 1948 (chairman)
Donald Munro, Suffolk	May 15, 1950
Albert A. Hornor, Suffolk	October 6, 1948
Frank R. Ober, Suffolk	October 6, 1948

Special Committees for 1950-1951

Elected by the Council, May 15, 1950

Cancer — Established 1947.

Ernest M. Daland, Suffolk, *chairman*; Thomas J. Anglem, Suffolk; Charles C. Lund, Suffolk; Channing C. Swan, Middlesex South; Shield Warren, Suffolk.

Hospital and Professional Relations — Established 1940.

Albert E. Parkhurst, Essex South, *chairman*; Joseph A. Holmes, Middlesex South; Storer P. Humphreys, Essex South; Arthur W. Allen, Suffolk; Donald A. Nickerson, Middlesex East; Leo V. Hand, Middlesex South; Richard B. Cattell, Suffolk.

Maternal Welfare — Established 1941.

Duncan Reid, Middlesex South, *chairman*; James M. Baty, Middlesex South; Arthur F. G. Edgelow, Hampden; Samuel Kirkwood, Middlesex East; Florence L. McKay, Suffolk; Louis E. Phaneuf, Suffolk; Luke Gillespie, Norfolk.

Postgraduate Medical Education — Established 1944.

Laurence B. Ellis, Suffolk, *chairman*; James Brosnan, Worcester; Harold F. Brown, Middlesex South; C. Nason Burden, Bristol North; Harwood Cummings, Franklin; John E. Dunphy, Norfolk; Alfred M. Glickman, Hampden; Francis J. Hanley, Plymouth; Eric Joslin, Berkshire; George P. Keaveny, Worcester North; Vlado A. Getting, Middlesex South; Lewis M. Hurxthal, Suffolk; John O. Niles, Barnstable; Robert H. Goodwin, Bristol South; Langdon Parsons, Middlesex South; Frederick R. Radcliffe, Essex North; Henry D. Stebbins, Essex South; Richard P. Stetson, Norfolk; Thomas J. G. Tighe, Middlesex North; W. Richard Ohler, Norfolk; Claude E. Welch, Middlesex South; Hilary F. White, Bristol South.

Medical Economics — Established 1944.

Arthur W. Allen, Suffolk, *chairman*; Vlado A. Getting, Middlesex South; Augustus Thorndike, Suffolk; Dean Clark, Suffolk; Norman A. Welch, Norfolk; Elmer S. Bagnall, Essex North.

To Make Recommendations as to Future Directors of Blue Shield — Established 1945.

Peirce H. Leavitt, Plymouth, *chairman* (term expires 1951); Elliott P. Joslin, Suffolk (term expires 1952); George G. Smith, Norfolk (term expires 1953); Thomas H. Lanman, Suffolk (term expires 1954); Albert E. Parkhurst, Essex South (term expires 1955).

To Meet with the Director of Veterans Administration Medical Care for Veterans and Their Dependents — Established 1945.

Humphrey L. McCarthy, Norfolk, *chairman*; James K. Bragger, Norfolk; Allen S. Johnson, Hampden.

Postgraduate Assembly — Established 1946.

Richard P. Stetson, Norfolk, *chairman*; Theodore L. Badger, Norfolk; Joseph S. Barr, Middlesex South; John F. Casey, Middlesex South; John F. Conlin, Suffolk; Laurence B. Ellis, Suffolk; Chester S. Keefer, Suffolk; John W. Norcross, Middlesex South; W. Richard Ohler, Norfolk; Leroy E. Parkins, Suffolk; Claude E. Welch, Middlesex South.

School Health — Established 1947.

Ernest M. Morris, Middlesex South, *chairman*; Stewart H. Clifford, Middlesex South; Allan R. Cunningham, Middlesex East; Reginald Fitz, Suffolk; Joseph Garland, Suffolk; Kenneth L. Maclachlan, Middlesex East; Thomas F. Reilly, Hampden.

Auditing — Established 1946.

Howard B. Jackson, Norfolk, *chairman*; Frank T. Downey, Middlesex South.

Advisory Council to Woman's Auxiliary — Established 1948.

John F. Conlin, Suffolk, *chairman*; David L. Belding, Norfolk South; Alfred M. Glickman, Hampden; Paul Jakmauh, Norfolk; Milton J. Quinn, Middlesex East.

Advisory Committee on Malpractice Insurance — Established 1948.

Carl Bearse, Norfolk, *chairman*; William J. Brickley, Suffolk; Maurice Fremont-Smith, Suffolk; Charles D. McCann, Plymouth; Horatio Rogers, Suffolk.

Emergency Medical Service — Established 1948.

Eugene C. Eppinger, Norfolk, *chairman*; Charles H. Bradford, Suffolk; Edward D. Churchill, Middlesex South; Donald E. Currier, Suffolk; J. Roswell Gallagher, Essex North; Allen S. Johnson, Hampden; James Jackson, Norfolk.*

Representatives to the Massachusetts Central Health Council.

Earle M. Chapman, Suffolk, *chairman*; Roy J. Ward, Worcester; Alfred L. Frechette, Norfolk; Alton S. Pope, Middlesex South; John F. Conlin, Suffolk.

Representative on a Professional Advisory Committee Organized by the Division of Vocational Rehabilitation of the State Department of Education for the Purpose of Establishing a Program of Physical Restoration.

Augustus Thorndike, Suffolk.

Representatives to the Council of the New England State Medical Societies.

William A. R. Chapin, Hampden; Gerald N. Hoeffel, Middlesex South; Norman A. Welch, Norfolk.

Twenty-Five Voting Members in Massachusetts Hospital Service, Inc.

Richard B. Butler, Bristol South; Laurence D. Chapin, Hampden; Lucien R. Chaput, Essex North; Henry W. Godfrey, Middlesex South; Harold H. Hamilton, Plymouth; Roy J. Heffernan, Norfolk; Albert A. Hornor, Suffolk; Harold R. Kurth, Essex North; Benjamin DeF. Lambert, Middlesex North; Alexander A. Levi, Middlesex South; Joseph C. Merriam, Middlesex South; Albert M. Moloney, Norfolk; Donald Munro, Suffolk; Donald A. Nickerson, Middlesex East; James P. O'Hare, Suffolk; Albert

*Since the meeting of the Council Dr. Eppinger has resigned as chairman of this Committee and Dr. Currier has been nominated as chairman with Dr. Bradford as co-chairman. Dr. Gallagher has resigned from the Committee and Dr. Richard Warren has been nominated to replace him.

E. Parkhurst, Essex South; Lewis S. Pilcher, Middlesex South; Helen S. Pittman, Suffolk; Allen G. Rice, Hampden; Laurence L. Robbins, Middlesex East; Arthur T. Ronan; Walter L. Sargent, Norfolk South; Monroe J. Schlesinger, Middlesex South; George L. Steele, Hampden; Sidney C. Wiggin, Suffolk.

Diabetes — Established 1949.

Howard F. Root, Suffolk, *chairman*; Frank N. Allan, Middlesex South; George Ballantyne, Worcester; Joseph Rosenthal, Norfolk; James H. Townsend, Middlesex South; Priscilla White, Suffolk; William M. Mason, Bristol South; Eoline DuBois, Hampden. Augustine Conroy, Middlesex North.

Veterans Administration Board of Review — Established 1949.

James K. Bragger, Norfolk, *chairman*; Edward P. Bagg, Hampden; John F. Conlin, Suffolk; Timothy F. P. Lyons, Norfolk; Humphrey L. McCarthy, Norfolk.

Rehabilitation and Chronic Disease — Established 1950.

Charles H. Bradford, Suffolk, *chairman*; Alexander P. Aitken, Middlesex East; Joseph S. Barr, Middlesex South; Walter Bauer, Suffolk; Hermann L. Blumgart, Suffolk; W. Irving Clark, Worcester; Otto J. Hermann, Norfolk; Arthur L. Watkins, Middlesex South, Augustus Thorndike, Suffolk; William Reggio, Suffolk; Robert Munroe, Norfolk; Robert Fleming, Norfolk.

Advisory Board — Established 1949.

Arthur W. Allen; Daniel B. Reardon; Edward P. Bagg; Dwight O'Hara; Reginald Fitz.

Chairman of the Blue Shield Fee Committee Chairmen — Established 1949.

Richard H. Sweet, Suffolk.

Committee on Fees — Established 1950.

Richard H. Sweet, Suffolk, *chairman*; George Prather, Norfolk; Leroy Schall, Middlesex South; William Beetham, Suffolk; Christopher Duncan, Suffolk; Theodore Badger, Norfolk; Samuel Robins, Norfolk; Leo V. Hand, Middlesex South; Richard I. Smith, Middlesex South; Donald Munro, Suffolk; William T. Green, Middlesex South.

Pharmaceutical Problems — Established 1950.

Frank N. Allan, Middlesex South, *chairman*; Charles L. Brennan, Middlesex North; George L. Maison, Norfolk.

Co-ordinating Committee — Established 1949.

Frank H. Lahey, Suffolk, *chairman*; Paul R. Hinchey, Essex South; Julius G. Kelley, Barnstable; Thurlow H. Pelton, Berkshire; Wm. J. Morse, Jr., Bristol North; Arthur F. Buckley, Bristol South; John J. D'Urso, Essex North; Lawrence R. Dame, Franklin; Samuel F. Potsubay, Hampden; Ralph S. Clapp, Hampshire; Kenneth L. Maclachlan, Middlesex East; Benjamin De F. Lambert, Middlesex North; Ivan G. Pyle, Middlesex South; Philip S. Foisie, Norfolk; William R. Helfrich, Norfolk South; Rudolph E. Swenson; Plymouth; Joseph A. Lundy, Worcester; Jesse C. Hales, Worcester North.

Pilot Clinics — Established 1949.

Brooks Ryder, Suffolk, *chairman*; Sidney Cobb, Worcester North; George R. Dunlop, Worcester; Alfred L. Frechette, Norfolk; David D. Rutstein, Norfolk.

Redistricting — Established 1949.

Charles G. Shedd, Suffolk, *chairman*; Alexander A. Levi, Middlesex South; Solomon L. Skvirsky, Norfolk; Donald Hight, Worcester; Milton M. Sisson, Franklin; Harold R. Kurth, Essex North; William F. Hickey, Jr., Middlesex East.

Supervision for the Red Cross Blood Program — Established 1950.

Lamar Soutter, Suffolk, *chairman*; F. Harold Allen, Jr., Middlesex South; Charles P. Emerson, Norfolk; Stephen Brown, Hampden; Harold B. Kenton, William C. Moloney, Norfolk; Dwight O'Hara, Middlesex South; Joseph F. Ross, Suffolk; Mark F. Lesses, Middlesex South; Carl W. Walter, Middlesex South; Stephen Maddock, Essex South; Loren D. Moore; Mario Stefanini.

Delegates and Alternates to the House of Delegates of the American Medical Association for 1950-1951

Delegates	Alternate
June 1, 1948, to December 31, 1950	
Patrick J. Sullivan, Berkshire	Nahum R. Pillsbury, Norfolk South
John J. Curley, Worcester North	Patrick E. Gear, Hampden
Walter G. Phippen, Essex South	John I. B. Vail, Barnstable
Frank W. Snow, Essex North	Frederick Hinchliffe, Norfolk South
Elected May 17, 1950, Term Begins January 1, 1951, to December 31, 1952.	
Charles G. Hayden, Middlesex South	Frank W. Snow, Essex North
Patrick J. Sullivan, Berkshire	Lawrence R. Dame, Franklin
James M. Faulkner, Norfolk	Lewis S. Pilcher Middlesex South
John J. Curley Worcester North	Donald R. Hayes, Hampden
Term expires December 31, 1951	
Charles J. Kickham, Norfolk	John Fallon, Worcester
Leland S. McKittrick, Suffolk	Harold R. Kurth, Essex North
Earle M. Chapman, Suffolk	Benjamin DeF. Lambert, Middlesex North

Delegates to the New England State Medical Societies

Maine	Samuel H. Proger, Norfolk
New Hampshire	Joseph Garland, Suffolk Storer P. Humphreys, Essex South
Vermont	Joseph Garland, Suffolk Sidney L. Morrison, Middlesex South
Rhode Island	William Mason, Bristol North Ernest M. Daland, Suffolk
Connecticut	Allen G. Rice, Hampden Thomas H. Lanman, Suffolk

Councilors for 1950-1951

Barnstable

Frank Travers, Barnstable, King's Highway, V. P.
F. O. Cass, Provincetown, 284A Commercial St.
A. J. D'Elia, Harwich Port, Main St., Leg. C.
D. E. Higgins, Cotuit, Main St., A. E. C., M. N. C.
H. P. Hopkins, Chatham, Main St., P. R. C.
S. L. Hunt, Yarmouth Port, Grand Army Highway, Sec.
H. F. Rowley, Harwich Port, E. C., A. M. N. C.
J. I. B. Vail, Hyannis, 155 Main St.

Berkshire

C. F. Kernan, Pittsfield, 184 North St., V. P.
D. N. Beers, Pittsfield, 74 North St., Sec.
W. A. Blais, Pittsfield, 74 North St.
R. J. Carpenter, North Adams, 85 Main St.
J. H. Fierman, Pittsfield, 74 North St., A. E. C., A. M. N. C.
T. W. Jones, Pittsfield, 18 Bank Row, Leg. C.

C. T. Leslie, Pittsfield, 18 Bank Row
T. H. Nelligan, Pittsfield, 184 North St.
Helen M. Scoville, Pittsfield, Pittsfield General Hospital, E. C., M. N. C.
P. J. Sullivan, Dalton, 471 Main St., P. R. C.

Bristol North

J. H. Brewster, Attleboro, 178 South Main St., V.P.
W. H. Bennett, Taunton, 79 Church Green
H. I. Bixby, Attleboro, 1st National Bank Building
C. N. Burden, Taunton, 3 Prospect St., Sec.
M. E. Johnson, Attleboro, 33 Bank St., P. R. C., A. E. C.
C. B. Kingsbury, Taunton, 63 Prospect St., A. M. N. C.
J. L. Murphy, Taunton, 23 Cedar St., M. N. C.
W. M. Stobbs, Attleboro, 63 Bank St., E. C., Leg. C.

Bristol South

J. P. Ponte, Jr., New Bedford, 202 Orchard St., V. P.
R. B. Butler, Fall River, 278 North Main St.
J. C. Corrigan, Fall River, 422 North Main St., A. E. C.
J. E. Fell, Fall River, 181 Purchase St., Sec.
D. F. Gallery, Fall River, 151 Rock St., Leg. C.
R. H. Goodwin, New Bedford, 15 South 6th St.
Herschel Heinz, New Bedford, 23 Maple St.
H. C. Lawson, Fall River, 124 Franklin St.
M. T. MacDonald, New Bedford, 99 Clinton St., P. R. C.
William Mason, Fall River, 151 Rock St., A. M. N. C.
D. R. Mills, Edgartown, Pease Point Way
H. E. Perry, New Bedford, 159 Cottage St., M. N. C.
A. J. Pothier, New Bedford, 720 County St.
C. C. Tripp, New Bedford, 416 County St., E. C.
Henry Wardle, Fall River, 173 Purchase St.

Essex North

F. C. Atkinson, Lawrence, 10 Amesbury St., V. P.
M. F. Ames, Newburyport, Box 88
E. S. Bagnall, Groveland, 281 Main St., Ex-Pres.
L. R. Chaput, Haverhill, 3 Washington Sq.
N. F. DeCesare, Lawrence, 57 Jackson St., Leg. C.
A. P. George, Haverhill, 32 Summer St., E. C.
H. R. Kurth, Lawrence, 57 Jackson St., Sec., P. R. C.
P. J. Look, Andover, 115 Main St., A. M. N. C.
L. C. Peirce, Newburyport, 279 High St.
F. R. Radcliffe, Haverhill, 116 Emerson St.
Nathan Silverman, Lawrence, 359 Broadway
F. W. Snow, Newburyport, 24 Essex St.
F. N. Sweetsir, Merrimac, 19 Main St., A. E. C.
C. F. Warren, Amesbury, 1 School St., M. N. C.
C. A. Weiss, Lawrence, 160 Jackson St.
J. F. Whitten, Amesbury, 448 Main St.

Essex South

S. N. Gardner, Salem, 24 Chestnut St., V. P.
Bernard Appel, Lynn, 281 Ocean St., P. R. C.
W. D. Babb, Salem, 40 Chestnut St.
W. W. Babson, Gloucester, 79 Prospect St.
K. J. Chadwell, Swampscott, 2 Fuller Ave.
D. S. Clark, Salem, 2 Oliver St., A. M. N. C.
Gerard Cote, Salem, 1 Harbor St.
J. J. Crowley, Lynn, 65 Broad St.
G. K. Fenn, Beverly, 8 Endicott St.
J. H. Fine, Beverly, 26 Abbott St.
T. H. Foote, Ipswich, 2 North Main St.
W. N. Goldberg, Danvers, 31 Conant St.
W. D. Higgins, Peabody, 6 Park St.
P. R. Hinchey, Salem, 355 Essex St., Leg. C.
F. P. Morse, Jr., Salem, 35 Summer St.
A. E. Parkhurst, Beverly, 1 Monument Sq., E. C.
M. H. Pett, Gloucester, 54 Middle St.
W. G. Phippen, Salem, 31 Chestnut St., Ex-Pres.
E. D. Reynolds, Danvers, 48 High St., A. E. C.
H. D. Stebbins, Salem, 342 Essex St., Sec.
C. F. Twomey, Lynn, 80 Ocean St., M. N. C.

Franklin

P. N. Freeman, Greenfield, 474 Main St., V. P.
J. R. Charron, Turners Falls, 60 Fourth St., A. E. C.
L. R. Dame, Greenfield, 78 Federal St., P. R. C.
F. W. Deane, East Northfield, 185 Main St., A. M. N. C.
H. R. Mahar, Orange, 1 High St., Leg. C.
J. E. Moran, Greenfield, 15 Franklin St., E. C.
M. M. Sisson, Greenfield, 31 Federal St., Sec.
W. D. Thomas, Montague, Central St., M. N. C.

Hampden

F. F. Celce, Holyoke, 190 Chestnut St., V. P.
M. S. Allan, Springfield, 127 Maple St.
E. P. Bagg, Holyoke, 207 Elm St., Ex-Pres.; A. M. N. C.; C.
R. L. Barrett, Springfield, 21 Maple St.
J. M. Birnie, Springfield, 146 Chestnut St., Ex-Pres.
M. J. Cariani, Springfield, 115 State St.
W. A. R. Chapin, Springfield, 121 Chestnut St.
J. L. Chereskin, Springfield, 333 Bridge St.
G. B. Corcoran, West Springfield, 84 Park St. A. E. C.
A. J. Douglas, Westfield, 30 Court St.
E. C. Dubois, Springfield, 20 Maple St.
Adolph Franz, Jr., Holyoke, 1158 Northampton St.
P. E. Gear, Holyoke, 188 Chestnut St.
J. M. Gilchrist, Springfield, 121 Chestnut St.
A. M. Glickman, Springfield, 283 Longhill St., Leg. C.
Frederic Hagler, Springfield, 20 Maple St., E. C.
D. R. Hayes, Springfield, 72 Harvard St.
H. P. Kennedy, Springfield, 115 State St.
John Pallo, Westfield, 97 Elm St.
S. F. Potsubay, Holyoke, 323 Appleton St.
L. A. Putnam, Holyoke, 399 Appleton St.
A. G. Rice, Springfield, 33 School St., Sec.
A. H. Riordan, Indian Orchard, 147 Oak St., P. R. C.
G. L. Schadt, Springfield, 44 Chestnut St., Ex-Pres.
J. A. Seaman, Springfield, 20 Maple St.
H. N. Simpson, Springfield, 276 Bridge St.
G. L. Steele, Springfield, 20 Maple St., M. N. C.

Hampshire

H. A. Tadgell, Belchertown, Belchertown State School, V. P.
R. S. Clapp, Amherst, 110 North Pleasant St.
J. J. Curran, Northampton, 16 Centre St., Leg. C.
J. R. Hobbs, Williamsburg, Main St., P. R. C., M. N. C., Sec.
M. T. Kennedy, Hadley, 11 Middle St., E. C.
J. G. Pekala, Northampton, 245 Main St.
L. B. Pond, Easthampton, 115 Main St., A. E. C., A. M. N. C.

Middlesex East

K. L. Maclachlan, Melrose, 1 Bellevue Ave., V. P.
C. R. Baisley, Reading, 46 Woburn St., Leg. C.
R. W. Daffinee, Melrose, 2 Porter St.
T. P. Devlin, Stoneham, 38 Pleasant St., E. C.
Robert Dutton, Wakefield, 33 Avon St., A. E. C.
W. F. Hickey, Jr., Winchester, 15 Dix St., Sec.
G. A. Marks, Winchester, 15 Dix St., P. R. C.
S. H. Moses, Winchester, 15 Dix St.
H. L. Mueller, Winchester, 31 Church St., A. M. N. C.
R. R. Stratton, Melrose, 538 Lynn Fells Parkway, C.
J. M. Wilcox, Woburn, 6 Bennett St., M. N. C.

Middlesex North

A. E. Conroy, Lowell, 45 Merrimack St., V. P.
P. G. Berman, Lowell, 174 Central St., Sec.
H. R. Coburn, Lowell, 202 Merrimack St., M. N. C.
W. M. Collins, Lowell, 174 Central St., A. M. N. C.
S. A. Dibbins, Lowell, 528 Andover St., P. R. C.
L. J. Hall, Lowell, 8 Merrimack St., A. E. C., Leg. C.
L. F. King, Lowell, 308 Merrimack St.
A. J. Stewart, Lowell, 310 Merrimack St., E. C.

Middlesex South

J. M. Baty, Belmont. Office, Brookline, 1101 Beacon St., V. P.
G. G. Bailey, Belmont. Office, Boston, 412 Beacon St.
E. W. Barron, Malden. Office, Boston, 20 Ash St.
Harris Bass, Everett, 351 Broadway
J. D. Bennett, West Somerville, 72 College Ave.
L. A. Blacklow, Belmont, 100 Leonard St.
W. O. Blanchard, Newton, 465 Centre St.
H. K. Bloom, Everett, 834 Broadway
G. F. H. Bowers, Newton Highlands, 156 Woodward St.
Alice M. Broadhurst, Watertown, 259 Mt. Auburn St., Leg. C.
Madelaine R. Brown, Cambridge. Office, Boston, 264 Beacon St.
R. N. Brown, Malden, 621 Main St.
R. W. Buck, Waban. Office, Boston, 5 Bay State Rd., C.

E. J. Butler, Cambridge, 25 Garden St.
J. F. Casey, Allston. Office, Boston, 475 Commonwealth Ave., A. E. C.
C. W. Clark, Newtonville, 363 Walnut St.
E. A. Cooney, Newton. Office, Boston, 270 Commonwealth Ave.
Oliver Cope, Cambridge. Office, Boston, Mass. General Hospital.
W. H. Crosby, Brighton, 304 Faneuil St.
H. J. Crumb, Lexington, 1632 Massachusetts Ave.
J. A. Daley, Natick, 36 Pond St.
Gordon Donaldson, Lincoln. Office, Boston, 266 Beacon St., C.
C. L. Derick, Newton Highlands. Office, Boston, 412 Beacon St.
J. G. Downing, Newton. Office, Boston, 520 Commonwealth Ave.
D. J. Duggan, Malden, 3 Hawthorne St.
A. G. Engelbach, Cambridge, 330 Mt. Auburn St.
W. C. Feeley, Cambridge, 859 Massachusetts Ave.
C. W. Finnerty, Somerville, 440 Broadway
J. M. Flynn, Belmont. Office, Boston, 412 Beacon St.
H. Q. Gallupe, Waltham. Office, Boston, 8 Fenway, Secretary
V. A. Getting, Belmont. Office, Boston, 546 State House
H. W. Godfrey, Auburndale, 14 Hancock St., A. M. N. C.
J. L. Golden, Medford, 86 Forest St.
A. D. Guthrie, Medford, 409 Salem St.
C. G. Hayden, Boston, 38 Chauncy St.
G. N. Hoeffel, Boston, 319 Longwood Ave.
J. A. Holmes, Waltham, 751 Main St.
W. T. Hood, Hudson, 20 Lincoln St.
Eliot Hubbard, Jr., Cambridge, 29 Highland St., Treasurer
H. W. Hudson, Jr., Brookline, 1101 Beacon St.
A. M. Jackson, Everett, 312 Broadway
F. R. Jouett, Cambridge, 1 Craigie St., M. N. C.
L. G. Kendall, Framingham, 198 Union Ave.
H. A. Kontoff, Newton Centre. Office, Boston, 479 Beacon St.
J. J. Lepore, Marlboro, 96 West Main St.
A. A. Levi, Newton. Office, Boston, 481 Beacon St., Sec.
H. E. MacMahon, Cambridge. Office, Boston, 416 Huntington Ave.
A. N. Makechnie, Cambridge, 14 Upland Rd.
R. A. McCarty, Waltham, 465 Lexington St.
J. H. McSweeney, Somerville, 26 Bow St.
J. C. Merriam, Framingham, 198 Union Ave., E. C.
Dudley Merrill, Cambridge, 51 Brattle St.
C. E. Mongan, Somerville, 24 Central St., Ex-Pres.
G. M. Morrison, Waban.Office, Boston, 520 Commonwealth Ave.
D. G. Nutter, Newton Centre, 1094 Centre St.
Dwight O'Hara, Waltham. Office, Boston, 136 Harrison Ave., Ex-Pres.
Fabyan Packard, Belmont, 154 Washington St.
L. S. Pilcher, Newton Centre. Office, Brookline, 1180 Beacon St., C.
Randolph Piper, Concord, 14 Sudbury Road
Max Ritvo, Newton. Office, Boston, 416 Marlboro St.
H. L. Robinson, West Newton, 80 Berkeley St.
L. G. Rondeau, Brighton, 555 Washington St.
G. A. Saunders, Arlington, 50 Pleasant St.
M. J. Schlesinger, Newton. Office, Boston, 330 Brookline Ave.
S. M. Simons, Arlington, 1250 Massachusetts Ave.
E. W. Small, Belmont, 68 Leonard St.
H. P. Stevens, Cambridge, 1 Craigie St.
A. B. Toppan, Watertown, 289 Mt. Auburn St.
J. H. Townsend, Cambridge, 330 Mt. Auburn St.
J. E. Vance, Natick. Office, Boston, 29 Bay State Rd.
C. F. Walcott, Cambridge, 81 Sparks St.
R. H. Wells, Lexington, 1430 Massachusetts Ave., P. R. C.
B. M. Wein, Newton. Office, Boston, 471 Commonweaith Ave.
Alfred Worcester, Waltham, 314 Bacon St., Ex-Pres.
Hovhannes Zovickian, Watertown, 528 Mt. Auburn St.

Norfolk

E. C. Smith, Brookline. Office, Boston, 520 Commonwealth Ave., V. P.
A. A. Abrams, Brookline, 1093 Beacon St.
C. E. Allard, Dorchester, 428 Columbia Rd.
B. E. Barton, West Roxbury, 10 Richwood St., E. C.
Carl Bearse, Boston, 483 Beacon St., M. N. C.
Elizabeth Broyles, Wellesley, Simpson Infirmary
J. H. Cauley, Dorchester, 8 Carruty St.
G. L. Doherty, West Roxbury. Office, Boston, 466 Commonwealth Ave.
A. L. Ehrenfried, Brookline. Office, Boston, 520 Beacon St.
J. M. Faulkner, Brookline. Office, Boston, 80 E. Concord St., C.
F. S. Foisie, Milton, 65 Hillsview Rd.
Susannah Friedman, Roxbury. Office, Boston, 485 Commonwealth Ave.
T. R. Goethals, Brookline, 34 Hawthorn Rd.
D. L. Halbersleben, Brookline, 42 Goodnough Rd.
H. B. Harris, Dorchester, 487 Columbia Rd.
R. J. Heffernan, Jamaica Plain. Office, Brookline, 1101 Beacon St.
Gilbert Horrax, Boston, 605 Commonwealth Ave.
P. J. Jakmauh, Milton. Office, South Boston, 300 Broadway, Leg. C.
I. R. Jankelson, Jamaica Plain. Office, Boston, 483 Beacon St.
L. F. Johnson, Brookline, Longwood Towers
C. J. Kickham, Brookline. Office, Boston, 508 Commonwealth Ave.
C. J. E. Kickham, Jamaica Plain. Office, Brookline, 1101 Beacon St.
D. L. Lionberger, Dedham, 709 East St.
D. S. Luce, Canton, 553 Washington St., P. R. C.
C. M. Lydon, Dorchester, 276 Bowdoin St.
D. L. Lynch, Roslindale, 61 Penfield St.
T. F. P. Lyons, Milton. Office, Boston, 270 Commonwealth Ave., A. E. C.
F. P. McCarthy, Milton. Office, Boston, 371 Commonwealth Ave.
H. L. McCarthy, Brookline. Office, Boston, 479 Beacon St.
R. T. Monroe, Brookline. Office, Boston, 270 Commonwealth Ave.
F. J. Moran, Dedham, 395 Washington St.
H. R. Morrison, Brookline. Office, Boston, 370 Marlboro St.
Hyman Morrison, Brookline. Office, Boston, 483 Beacon St.
D. J. Mullane, Brookline, 1101 Beacon St.
H. A. Novack, Brookline. Office, Boston, 471 Commonwealth Ave.
J. J. O'Connell, Dorchester, 1061 Dorchester Ave.
W. R. Ohler, Jamaica Plain. Office, Boston, 319 Longwood Ave.
E. E. O'Neil, Brookline. Office, Boston, 270 Commonwealth Ave.
R. S. Palmer, Brookline. Office, Boston, 330 Dartmouth St.
G. W. Papen, Brookline. Office, Boston, 31 Milk St.
H. C. Petterson, West Roxbury. Office, Boston, 29 Bay State Rd.
S. H. Proger, Brookline. Office, Boston, 30 Bennet St.
H. A. Rice, Canton, 472 Washington St., A. M. N. C.
S. A. Robins, Boston, 636 Beacon St.
D. D. Scannell, Jamaica Plain. Office, Boston, 475 Commonwealth Ave.
J. A. Seth, Milton. Office, Boston, 47 Bay State Rd.
L. A. Sieracki, Norwood, 71 Winter St.
S. L. Skvirsky, Brighton. Office, Boston, 545 State House, Sec.
Kathleyne S. Snow, Jamaica Plain. Office, Boston, 466 Commonwealth Ave.
J. W. Spellman, Chestnut Hill. Office, Brookline, 1101 Beacon St.
A. R. Stagg, Medfield, 25 Pleasant St.
W. J. Walton, Dorchester, 106 Bowdoin St.
N. A. Welch, West Roxbury. Office, Boston, 520 Commonwealth Ave., Assistant Treasurer
W. A. White, Jr., Milton. Office, Boston, 270 Commonwealth Ave.
G. F. Wilkins, Brookline. Office, Boston, 245 State St.
F. R. Withington, Milton, 350 Randolph Ave.
Marjorie Woodman, Jamaica Plain. Office, Boston, 21 Bay State Rd.
E. T. Wyman, Brookline. Office, Boston, 319 Longwood Ave.

Norfolk South

R. G. Vinal, Norwell, Main St., V. P.
F. A. Bartlett, Wollaston, 308 Beale St.
D. L. Belding, Hingham, 215 Main St., Leg. C.
R. L. Cook, Quincy, 1245 Hancock St., E. C., M. N. C
E. B. Fitzgerald, Wollaston, 563 Furnace Brook Parkway
W. R. Helfrich, Quincy, 17 Whitney Rd., A. E. C., A. M. N. C.
Frederick Hinchliffe, Cohasset, 117 South Main St.
H. F. Howe, Cohasset, 11 North Main St.
E. K. Jenkins, South Braintree, Norfolk County Hosp., Sec.
C. J. Lynch, Quincy, 25 Elm St.
N. R. Pillsbury, South Braintree, Norfolk County Hospital
D. B. Reardon, Quincy, 1186 Hancock St., P. R. C., Ex-Pres.
Morgan Sargent, Quincy, 24 Whitney Road

Plymouth

A. L. Duncombe, Brockton, 38 Winthrop St., V. P.
J. C. Angley, Bryantville, School St., P. R. C.
J. M. Bonnar, Jr., Middleboro, 33 Pearl St.
Hyman Duby, Plymouth, 345 Court St.
Samuel Gale, Brockton, 12 Cottage St., Sec.
H. H. Hamilton, Plymouth, 70 Court St., Leg. C.
F. J. Hanley, Brockton, 1091 North Main St.
A. L. Hurlburt, East Bridgewater, 81 Central St., A. E. C.
P. H. Leavitt, Brockton, 129 West Elm St., M. N. C.
D. A. Martin, South Hanson, Plymouth County Hosp., A. M. N. C.
C. D. McCann, Brockton, 12 Cottage St., E. C.
G. A. Moore, Brockton, 167 Newbury St.
R. E. Swenson, Plymouth, 1 Carver St.

Suffolk

L. J. McCarthy, Chelsea. Office, Boston, 520 Commonwealth Ave., V. P.
H. L. Albright, Boston, 412 Beacon St.
A. W. Allen, Boston, 266 Beacon St., Ex-Pres.
T. J. Anglem, Brookline, 1180 Beacon St.
J. W. Bartol, Boston, 1 Chestnut St., Ex-Pres.
C. H. Bradford, Boston, 520 Commonwealth Ave.
W. J. Brickley, Boston, 524 Commonwealth Ave.
W. E. Browne, Boston, 587 Beacon St., Leg. C., A. E. C.
A. M. Butler, Boston, Mass. General Hospital
A. J. A. Campbell, Brighton. Office, Boston, 520 Commonwealth Ave.
E. M. Chapman, Boston, 266 Beacon St.
M. H. Clifford, Boston, 501 Boylston St.
J. F. Collins, Revere, 123 Bennington St.
J. F. Conlin, Boston, 8 Fenway
A. P. DerHagopian, Chelsea, 39 Cary Ave.
E. A. Edwards, Boston, 372 Marlboro St.
L. B. Ellis, Cambridge. Office, Boston, 319 Longwood Ave.
Reginald Fitz, Boston, 251 Longwood Ave., Ex-Pres.
Maurice Fremont-Smith, Boston, 12 Hereford St.
Channing Frothingham, Boston, 101 Bay State Rd., Ex-Pres.
Joseph Garland, Brookline. Office, Boston, 8 Fenway
G. L. Gately, East Boston, 624 Bennington St., P. R. C.
R. L. Goodale, Cambridge. Office, Boston, 330 Dartmouth St.
A. A. Hornor, Brookline. Office, Boston, 319 Longwood Ave.
L. M. Hurxthal, West Newton. Office, Boston, 605 Commonwealth Ave.
H. A. Kelly, Winthrop, 200 Pleasant St., E. C.
H. E. Kennard, Brookline, 1180 Beacon St.
F. H. Lahey, Boston, 605 Commonwealth Ave.
T. H. Lanman, Chestnut Hill. Office, Boston, 300 Longwood Ave.
R. I. Lee, Boston, 264 Beacon St., Ex-Pres.
C. C. Lund, Boston, 20 Gloucester St., A. M. N. C.
C. F. Maraldi, Boston, 276 Commonwealth Ave.
H. C. Marble, Newton Centre. Office, Boston, 270 Commonwealth Ave.
L. S. McKittrick, Brookline, 1180 Beacon St., President
Charles Meloni, East Boston, 305 Havre St.
Donald Munro, Boston, 818 Harrison Ave.
H. L. Musgrave, Revere, 620 Beach St.
F. R. Ober, Boston, 234 Marlboro St., C., Ex-Pres.
D. J. O'Brien, Winthrop, 78 Washington St.
J. P. O'Hare, Chestnut Hill. Office, Boston, 520 Commonwealth Ave.
L. E. Parkins, Brookline. Office, Boston, 12 Bay State Rd.
Helen S. Pittman, Boston, 264 Beacon St.
J. J. Regan, South Boston. Office, Boston, 520 Commonwealth Ave.
W. H. Robey, Boston, 202 Commonwealth Ave., Ex-Pres.
Horatio Rogers, Boston, 264 Beacon St., C.
H. F. Root, Brookline. Office, Boston, 81 Bay State Rd.
Weston Sewall, Roslindale. Office, Boston, 466 Commonwealth Ave.
C. G. Shedd, Wellesley. Office, Brookline, 1180 Beacon Street, Sec.
R. M. Smith, Boston, 330 Dartmouth St., C.
Lendon Snedeker, Brookline. Office, Boston, 300 Longwood Ave.
C. M. Stearns, Chelsea, 116 Hawthorn St.
Conrad Wesselhoeft, Boston, 315 Marlboro St., M. N. C.
S. C. Wiggin, Waban. Office, Boston, 270 Commonwealth Ave.

Worcester

Thomas Hunter, Shrewsbury, 552 Main St., V. P.
C. E. Ayers, Worcester, 36 Pleasant St.
G. W. Ballantyne, Worcester, 27 Elm St., A. M. N. C.
Jacob Brem, Worcester, 796 Pleasant St.
Edward Budnitz, Worcester, 44 Cedar St., Sec.
J. B. Butts, Worcester, 24 Franklin St., Leg. C.
J. T. B. Carmody, Worcester, 340 Main St.
F. B. Carr, Worcester, 27 Elm St., M. N. C.
E. J. Crane, Holden, Armington Lane
Paul Dufault, Rutland, Rutland State Sanatorium
G. R. Dunlop, Worcester, 27 Elm St.
W. J. Elliott, Worcester, 119 Belmont St.
John Fallon, Worcester, 10 Institute Rd., E. C.
L. M. Felton, Worcester, 36 Pleasant St.
C. V. King, Worcester, 47 Boyd St.
H. L. Kirkendall, Worcester, 27 Elm St.
D. G. Ljungberg, Worcester, 36 Pleasant St.
J. A. Lundy, Worcester, 16 Norwich St.
G. E. McCabe, Worcester, 773 Park Ave.
J. C. McCann, Worcester, 390 Main St.
D. K. McClusky, Worcester, 7 Hawthorne St.
J. W. McKoan, Worcester, 36 Pleasant St.
J. M. Olson, Westboro, 54 West Main St.
F. A. O'Toole, Clinton, 101 Chestnut St.
E. L. Richmond, Worcester, 390 Main St.
N. S. Scarcello, Worcester, 1 Selden St., A. E. C., P. R. C.
J. J. Tegelberg, Worcester, 390 Main St.
R. J. Ward, Worcester, 9 Bellevue St., C.
B. C. Wheeler, Worcester, 27 Elm St.

Worcester North

J. J. Curley, Leominster, 89 West St., V. P.
D. B. Cheetham, Athol, 164 Exchange St., P. R. C.
K. J. Jolma, Gardner, 86 Jonathan St., A. E. C.
J. P. Marnane, Gardner, 4 Comee St., Leg. C.
J. V. McHugh, Leominster, 55 West St., E. C., M. N. C.
S. I. Nathanson, Fitchburg, 882 Main St., Sec.
J. G. Simmons, Fitchburg, 30 Myrtle Ave., A. M. N. C.
H. J. Sparling, Fitchburg, 46 Merriam Parkway

The initials *E. C.* following the name of a Councilor indicate that he is a member of the Executive Committee, and *A. E. C.* that he is an alternate member of the Executive Committee; *M. N. C.* that he is a member of the Committee on Nominations and *A. M. N. C.* that he is an alternate member of the Committee on Nominations; *Leg. C.* that he is a member of the Committee on Legislation; *P. R. C.* that he is a member of the Committee on Public Relations; *V. P.* that a member is a councilor by virtue of his office as president of a district society and so vice-president of the general society; *C.* by virtue of his office as chairman of a standing committee; *Sec.* by virtue of his office as secretary of a district society and *Ex-Pres.* by virtue of being a past president.

Censors for 1950-1951

Barnstable
D. E. Higgins, Cotuit, *supervisor.*
D. H. Hiebert, Provincetown.
Oscar S. Simpson, Centerville.
Joseph Kelly, Orleans.
J. L. Chute, Osterville.

Berkshire
C. T. Leslie, Pittsfield, *supervisor.*
M. A. Gangemi, North Adams.
N. N. Copeland, Pittsfield.
T. H. Nelligan, Pittsfield.
G. S. Wickham, Lee.

Bristol North
J. L. Murphy, Taunton, *supervisor.*
H. G. Vaughan, Attleboro.
C. B. Kingsbury, Taunton.
A. J. Leddy, Taunton.
J. H. Brewster, Attleboro.

Bristol South
Herschel Heinz, New Bedford, *supervisor.*
E. L. Merritt, Fall River.
Samuel Brown, Fall River.
W. F. McKnight, Fall River.
H. P. Harney, New Bedford.

Essex North
L. C. Peirce, Newburyport, *supervisor.*
R. J. Neil, Methuen.
J. A. Sapienza, Lawrence.
Frank Schlosberg, Haverhill.
David Zelig, Haverhill.

Essex South
S. N. Gardner, Salem, *supervisor.*
W. R. Irving, Gloucester.
H. J. Oberson, Lynn.
E. D. Reynolds, Danvers.
J. R. Shaughnessy, Salem.

Franklin
John Moran, Greenfield, *supervisor.*
A. E. Johnson, Deerfield.
Henry Rys, Turners Falls.
Edwin Thorn, Greenfield.
F. J. Barnard, Greenfield.
Warren Thomas, Turners Falls.

Hampden
John Pallo, Westfield, *supervisor.*
E. W. Beauchamp, Springfield.
L. A. Putnam, Holyoke.
A. A. Palermo, Springfield.
J. L. Smead, Springfield.

Hamp shire
L. B. Pond, Easthampton, *supervisor.*
Stephen Brown, Northampton.
T. F. Corriden, Northampton.
M. I. Cooney, Northampton.
J. E. Hayes, Northampton.

Middlesex East
S. H. Moses, Winchester, *supervisor.*
H. A. Bouve, Wakefield.
M. J. Klainer, Stoneham.
R. E. Militzer, Woburn.
A. E. Morris, Reading.

Middlesex North
Leo King, Lowell, *supervisor.*
George Duggan, Lowell.
David Latham, Lowell.
Philip Mullane, Lowell.
Walter Twarog, Lowell.

Middlesex South
H. J. Crumb, Lexington, *supervisor.*
E. A. Gaston, Framingham.
W. O. Blanchard, Newton.
Dudley Merrill, Cambridge.
H. K. Bloom, Everett.

Norfolk
K. S. Snow, Jamaica Plain, *supervisor.*
G. L. Doherty, West Roxbury.
W. C. Moloney, Jamaica Plain.
I. R. Jankelson, Brookline.
H. A. Novack, Brookline.

Norfolk South
F. A. Bartlett, Wollaston, *supervisor.*
Arthur Rappaport, Quincy.
H. S. Reid, Cohasset.
R. E. Ross, Braintree.
W. L. Sargent, Quincy.

Plymouth
R. E. Swenson, Plymouth, *supervisor.*
G. A. Buckley, Brockton.
W. G. Gould, Kingston.
D. W. Pope, Brockton.
C. D. McCann, Brockton.

Suffolk
L. M. Hurxthal, Boston, *supervisor.*
J. F. Collins, Revere.
J. J. Todd, Boston.
T. J. Anglem, Brookline.
C. F. Maraldi, Boston.

Worcester
J. W. McKoan, Jr., Worcester, *supervisor.*
E. L. Richmond, Worcester.
H. L. Kirkendall, Worcester.
E. J. Croce, Worcester.
Sanfrey Lilyestrom, Worcester.

Worcester North
K. K. Jolma, Gardner, *supervisor.*
E. R. Pickwick, Fitchburg.
F. X. Dufault, Athol.
C. A. Wheeler, Leominster.
F. R. Pierce, Gardner.

Vice-Presidents of the Massachusetts Medical Society (*Ex-Officiis*) for 1950-1951

Presidents of District Medical Societies

(Arranged according to seniority of fellowship in the Massachusetts Medical Society)

Hampden — Frederick F. Celce, Holyoke.
Essex North — Frederick C. Atkinson, North Andover.
Bristol South — J. P. Ponte, Jr., New Bedford.
Worcester North — John J. Curley, Leominster.
Middlesex North — Augustine E. Conroy, Lowell.
Essex South — Stuart N. Gardner, Salem.
Bristol North — James H. Brewster, Attleboro.
Middlesex East — Kenneth L. Maclachlan, Melrose.
Berkshire — Clement F. Kernan, Pittsfield.
Hampshire — Henry A. Tadgell, Belchertown.
Middlesex South — James Marvin Baty, Belmont.
Franklin — Perrin N. Freeman, Greenfield.
Suffolk — Lawrence J. McCarthy, Boston.
Worcester — Thomas Hunter, Shrewsbury.
Barnstable — Frank Travers, Barnstable.
Norfolk — Edward C. Smith, Brookline.
Plymouth — A. L. Duncombe, Brockton.
Norfolk South — Raymond G. Vinal, Norwell.

Commissioners of Trial for 1950-1951

Barnstable — Frank O. Cass, Provincetown.
Berkshire — James W. Bunce, North Adams.
Bristol North — John W. Cook, Mansfield.

Bristol South — Arthur C. Lewis, Fall River.
Essex North — Harry H. Nevers, Lawrence.
Essex South — Thomas B. Rafferty, Lynn.
Franklin — Kenneth Jacobus, Turners Falls.
Hampden — George D. Henderson, Holyoke.
Hampshire — Robert C. Byrne, Hatsfield.
Middlesex East — George R. Murphy, Melrose.
Middlesex North — Frank R. Brady, Lowell.
Middlesex South — Horace P. Stevens, Cambridge.
Norfolk — William J. Walton, Dorchester.
Norfolk South — George D. Dalton, Quincy.
Plymouth — E. L. Perry, Middleboro.
Suffolk — Frederick W. O'Brien, Boston.
Worcester — Charles V. King, Worcester.
Worcester North — F. Richard Pierce, Gardner.

Officers of the Sections for 1950-1951

Medicine

Chairman, Frank B. Carr, Worcester; *vice-chairman*, Donald S. King, Boston; *secretary*, Joseph Frothingham, Boston.

Physical Medicine

Chairman, Volta Hall, Boston; *secretary*, Sidney Licht, Boston.

Dermatology

Chairman, Loretta J. Cummins, Boston; *secretary*, Paul J. Catinella, Boston.

Radiology

Chairman, Magnus I. Smedal, Waban; *secretary*, John Turner, Springfield.

Obstetrics and Gynecology

Chairman, Duncan E. Reid, Boston; *vice-chairman*, Arthur J. Gorman, Brookline; *secretary*, Joseph F. Ferrone, Newton.

Industrial Health

Chairman, Daniel L. Lynch, Roslindale; *secretary*, Karl T. Benedict, West Boylston.

Psychiatry and Neurology

Chairman, Augustus S. Rose, Boston; *secretary*, John T. Shea, Foxboro.

Surgery

Chairman, J. Hartwell Harrison, Brookline; *secretary*, Lee G. Kendall, Framingham. Executive Committee — Richard H. Thompson, Salem, and George R. Dunlop, Worcester.

Pediatrics

Chairman, R. Cannon Eley, Boston; *secretary*, Harry L. Mueller, Winchester.

Physiology and Pathology

Chairman, Robert Fienberg, Framingham; *secretary*, Stanley L. Robbins, Brookline.

Anesthesiology

Chairman, Leo V. Hand, Boston; *secretary*, Philip S. Marcus, Boston.

Ophthalmology and Otolaryngology

Chairman, James J. Regan, South Boston; *vice-chairman*, Leroy A. Schall, Boston; *secretary*, Lawrence R. Dame, Greenfield.

Officers of the District Medical Societies for 1950-1951

Barnstable — *President*, Frank Travers, Barnstable; *vice-president*, Donald E. Higgins, Cotuit; *secretary*, Sheldon L. Hunt, Yarmouth Port; *treasurer*, John O. Niles, Osterville; *librarian*, Carroll H. Keene, Chatham; *executive councilor*, Harold F. Rowley, Harwich Port; *public relations councilor*, Henry P. Hopkins, Chatham; *legislative councilor*, Arthur J. D'Elia.

Berkshire — *President*, Clement Kernan, Pittsfield; *vice-president*, Garvey Adeson, Pittsfield; *secretary*, Daniel N. Beers, Pittsfield; *treasurer*, Theodore W. Jones, Pittsfield; *executive councilor*; Helen M. Scoville, Pittsfield; *public relations councilor*, Patrick J. Sullivan, Dalton; *legislative councilor*, Theodore W. Jones, Pittsfield.

Bristol North — *President*, James H. Brewster, Attleboro; *vice-president*, Curtis B. Kingsbury, Taunton; *secretary*, C. Nason Burden, Taunton; *treasurer*, Charles E. Hoye, Jr., Taunton; *executive councilor*, William M. Stobbs, Attleboro; *public relations councilor*; Milton E. Johnson, Attleboro; *legislative councilor*, William M. Stobbs, Attleboro.

Bristol South — *President*, Joseph P. Ponte, Jr., New Bedford; *vice-president*, William Mason, Fall River; *secretary-treasurer*, James E. Fell, Fall River; *executive councilor*, Curtis C. Tripp, New Bedford; *public relations councilor*, Milton T. MacDonald; *legislative councilor*, Daneil F. Gallery, Fall River.

Essex North — *President*, Frederick C. Atkinson, North Andover; *vice-president*, Herbert A. Fenton, Lawrence; *secretary*, Harold R. Kurth, Lawrence; *treasurer*, J. LeRoy Wood, Lawrence; *librarian*, Arthur W. Clancy, Newburyport; *executive councilor*, Arnold P. George, Haverhill; *public relations councilor*, Harold R. Kurth, Lawrence; *legislative councilor*, Lucien R. Chaput, Haverhill.

Essex South — *President*, Stuart N. Gardner, Salem; *vice-president*, William W. Babson, Gloucester; *secretary*, Henry D. Stebbins, Marblehead; *treasurer*, Andrew Nichols, III, Danvers; *executive councilor*, Albert E. Parkhurst, Beverly; *public relations*, Bernard Appel, Lynn; *legislative councilor*, Paul R. Hinchey, Salem.

Franklin — *President*, Perrin Freeman, Greenfield; *vice-president*, Harwood Cummings, Greenfield; *secretary-treasurer*, Milton M. Sisson, Greenfield; *executive councilor*, John Moran, Greenfield; *public relations councilor*, Lawrence R. Dame, Greenfield; *legislative councilor*, Harold Mahar, Orange.

Hampden — *President*, Frederick W. Celce, Holyoke; *vice-president*, James L. Smead, Springfield, *secretary-treasurer*, Allen G. Rice, Springfield; *executive councilor*, Frederic Hagler, Springfield; *public relations councilor*, Arthur H. Riordan, Springfield; *legislative councilor*, Alfred M. Glickman, Springfield.

Hampshire — *President*, Henry A. Tadgell, Belchertown; *vice-president*, Joseph G. Pekala, Northampton; *secretary-treasurer*, Joseph R. Hobbs, Williamsburg; *librarian*, Frances C. McInnes, Northampton; *executive councilor*, Maurice T. Kennedy, Hadley; *public relations councilor*, Joseph R. Hobbs, Williamsburg; *legislative councilor*, John J. Curran, Northampton.

Middlesex East — *President*, Kenneth L. Maclachlan, Melroae; *vice-president*, Ernest C. MacDougall, Wilmington; *secretary*, William F. Hickey, Jr., Winchester; *treasurer*, Charles W. DeWolf, Wakefield; *librarian*, Angelo L. Maietta, Winchester; *executive councilor*, Thomas P. Devlin, Stoneham; *public relations councilor*, George A. Marks, Winchester; *legislative councilor*, Charles R. Baisley, Reading.

Middlesex North — *President*, Augustine Conroy, Lowell; *vice-president*, Harold Leland, Lowell; *secretary*, Philip G. Berman, Lowell; *treasurer*, Mason D Bryant, Lowell; *librarian*, Anna Q. Churchill, Lowell; *executive councilor*, Artemas Stewart, Lowell; *public relations councilor*, Samuel Dibbins, Lowell; *legislative councilor*, Leonard Hall, Lowell.

Middlesex South — *President*, James Marvin Baty, Belmont; *vice-president*, Joseph C. Merriam, Framingham; *secretary*, Alexander A. Levi, Newton; *treasurer*, Fabyan Packard, Belmont; *executive councilor*, Joseph C. Merriam, Framingham; *public relations councilor*, Ralph H. Wells, Lexington; *legislative councilor*, Alice M. Broadhurst, Watertown.

Norfolk — *President*, Edward C. Smith, Brookline; *vice-president*, Philip S. Foisie, Milton; *secretary*, Solomon L. Skvirsky, Brighton; *treasurer*, Archie A. Abrams, Brookline; *executive councilor*, Basil E. Barton, West Roxbury; *public relations councilor*, Dean S. Luce, Canton; *legislative councilor*, Paul J. Jakmauh, Milton.

Norfolk South — *President*, Raymond G. Vinal, Norwell; *vice-president*, Cornelius J. Lynch, Quincy; *secretary*, Ebenezer K. Jenkins, Braintree; *executive councilor*, Robert L. Cook, Quincy; *public relations councilor*, Daniel B. Reardon, Quincy; *legislative councilor*, David L. Belding, Hingham.

Plymouth — *President*, A. L. Duncombe, Brockton; *vice-president*, Fred F. Weiner, Brockton; *secretary*, Samuel Gale, Brockton; *treasurer*, Henry Rabnowitz, Brockton; *librarian*, John A. Carriuolo, Brockton; *executive councilor*, Charles McCann, Brockton; *public relations councilor*, John C. Angley, Bryantville; *legislative councilor*, H. H. Hamilton, Plymouth.

Suffolk — *President*, Lawrence J. McCarthy, Boston; *vice-president*, Charles H. Bradford, Boston; *secretary*, Charles G. Shedd, Brookline; *treasurer*, Sidney C. Wiggin, Boston; *executive councilor*, Harvey A. Kelly, Winthrop; *public relations councilor*, G. Lynde Gately, East Boston; *legislative councilor*, William E. Browne, Boston.

Worcester — *President*, Thomas Hunter, Shrewsbury; *vice-president*, Charles E. Ayers, Worcester; *secretary*, Edward Budnitz, Worcester; *treasurer*, Arthur D. Ward, Worcester; *executive councilor*, John Fallon, Worcester; *public relations councilor*, Nicholas S. Scarcello, Worcester; *legislative councilor*, John B. Butts, Worcester.

Worcester North — *President*, John J. Curley, Leominster; *vice-president*, Joseph A. McLean, Ayer; *secretary*, Seymour I. Nathanson, Fitchburg; *treasurer* Frederick H. Thompson, Jr., Fitchburg; *executive councilor*, James V. McHugh, Leominster; *public relations councilor*, Donald B. Cheetham, Athol; *legislative councilor*, Joseph P. Marnane, Gardner.

Admissions Recorded from May 23, 1949, to May 16, 1950

YEAR OF ADMISSION	NAME AND RESIDENCE	MEDICAL SCHOOL
1950	Abelman, Lester M., Allston	Tufts
1950	Adelson, Lester, Boston	Tufts
1949	Alicandri, B. Bruce, Springfield	New York Medical College
1950	Allain, Donald, Worcester	Boston University
1949	Almeida, Victor, New Bedford	Tufts
1949	Aloia, Anthony Francis, Leominster	Boston University
1950	Ambadgis, Theodore T., Brookline	Boston University
1949	Anderson, Paul O., Rutland Heights	Boston University
1949	Angeles, Triantaphyllos A., Haverhill	Middlesex
1950	Appleby, Leonard W., Brookline	McGill University
1949	Armstrong, Stewart, Brookline	New York University
1949	Arragg, Fred George, Lawrence	Tufts
1950	Atkinson, Edward K., Brighton	University of Pennsylvania
1949	Baer, Arthur Edward, Ispwich	Middlesex
1950	Banta, Edwin V., Lawrence	University of Michigan
1950	Baum, Marvin, Worcester	New York University
1950	Baum, Stanley M., Springfield	Middlesex
1950	Beach, Carroll C., Jr., Worcester	New York University
1949	Becker, Merrill Martin, Springfield	St. Louis University
1949	Bering, Edgar A., Jr., Boston	Harvard
1949	Berlad, Milton, Scituate Harbor	Middlesex
1949	Beroz, Ernest, Lawrence	Middlesex
1950	Berry, H. Meredith, Brockton	Cornell University
1950	Berry, John L., Pittsfield	University of Vermont
1950	Bevilacqua, Rocco P., Wellesley	Long Island College of Medicine
1950	Blackburn, Charles F., Framingham	Tufts
1949	Blazar, Howard A., Boston	Columbia
1950	Bockoven, John S., Boston	University of Vermont
1949	Boog, Janet Margaret, Lawrence	New York Medical College
1950	Breit, George B., Becket	Middlesex
1949	Brewer, Robert DuBois, Jr., Gloucester	New York University
1950	Brown, Hathorn P., Boston	Harvard
1950	Brown, Josiah W., Newton	Long Island College of Medicine
1950	Brown, Thornton, Boston	Harvard
1949	Bryant, Carroll, Springfield	Tufts
1949	Buckley, Arthur Francis, New Bedford	Tufts
1950	Buckner, Louise P., Northampton	University of Maryland
1949	Buka, Robert, Jr., Boston	University of Pittsburgh
1950	Bullard, John C., Dover	Harvard
1950	Bullowa, Margaret, Boston	New York University
1949	Burns, John Joseph, Brookline	Tufts
1950	Burwell, Stanley W., Boston	Columbia
1950	Bush, Richard D., Philadelphia	Johns Hopkins University
1950	Bush, Robert T., Brimfield	Tufts
1949	Caldwell, Renwick Keese, Northampton	University of Vermont
1949	Campbell, Donald Ignatius, Weston	Dalhousie University
1949	Capers, Raymond, Lowell	Middlesex
1950	Carr, Edward C., Somerville	Middlesex
1950	Carrigan, Walter E., Malden	Tufts
1949	Casserly, George Bartholomew, Fall River	Tufts
1950	Caton, William L., Jr., Boston	Washington University
1949	Cenni, Louis J., Worcester	Temple University
1949	Chrisman, Oscar Donald, Northampton	Harvard

1949	Church, Richard E., Millbury	Tufts
1949	Churchville, Lawrence A., Townsend	Kansas City University
1950	Clark, Dean A., Boston	Johns Hopkins
1949	Cleveland, Harold Frank, Springfield	Middlesex
1950	Coggan, Manuel, Lowell	Middlesex
1949	Cohen, Albert, Cambridge	Tufts
1950	Cohen, Felix, Brookline	University of Rochester
1949	Collard, Kenneth L., Belchertown	Tufts
1949	Colpoys, Francis L., Jr., Framingham	Tufts
1949	Cooperstein, Menahem, Taunton	Boston University
1949	Corbin, George C., Boston	Howard University
1949	Corcoran, James, Boston	Columbia
1950	Cordaro, Benjamin J., Holbrook	College of Physiciand and Surgeons, Boston
1949	Cosgrove, Thomas C., Vineyard Haven	College of Physicians and Surgeons, Boston
1950	Costas, Charles W., Peabody	Kansas City University
1950	Craige, Ernest, Boston	Harvard
1950	Cressy, Norman L., Northampton	Yale
1950	Crocker, Augustus T., Boston	Tufts
1949	Crocker, Harvey J., Essex	Tufts
1950	Crocker, Seth C., Framingham	Harvard
1950	Curtis, David M., Hyannis	Tufts
1950	Curtiss, David P., Williamstown	Long Island College of Medicine
1950	Cutler, Herbert, Allston	Middlesex
1950	Daley, John M., Wellesley	Columbia University
1949	Dalton, John T., Braintree	Tufts
1950	DeFriez, Albert I. C., Chestnut Hill	Columbia University
1950	Delery, Alfred A., Medford	Tufts
1950	Derrick, William S., Boston	George Washington University
1949	Dervan, Edward J., Newburyport	Boston University
1949	DiCicco, John P., Webster	Middlesex
1950	DiStasio, Guy A., Revere	Middlesex
1950	Donovan, Thomas M., Fitchburg	Tufts
1949	Douglas, Richard K., Westfield	Albany Medical College
1950	Dreyfus, John, Quincy	Middlesex
1949	Dreyfus, Joseph E., Mattapan	Middlesex
1950	Drinan, Francis W., West Newton	Tufts
1950	Dunn, John F., Jr., Worcester	Tufts
1949	Dunstan, Paul L., Waverley	Baylor University
1950	Durant, John E. C., New Bedford	Tufts
1950	Dworkin, Samuel S., Adams	Long Island College of Medicine
1949	Elkin, Milton, West Roxbury	Harvard
1949	Ellis, Victor, Alston	Middlesex
1950	Evans, Henry D., Natick	Middlesex
1950	Eyler, William R., Boston	Harvard
1949	Ferrera, Charles John, Chelsea	Middlesex
1950	Ferrino, Peter J., East Boston	Kansas City University
1949	Finck, Albert Jacob, West Roxbury	Harvard
1950	Fisher, James D., Springfield	University of Cincinnati
1950	Fontneau, Nelson C., Winchester	Harvard
1949	Forster, Robert E., II, Boston	University of Pennsylvania
1949	Foster, John Tallmadge, Medford	Jefferson Medical College
1949	Fowler, Dwight Marshall, Framingham	Harvard
1949	Frank, Edward David, Boston	New York University
1950	Franklin, Stanley H., Brookline	Middlesex
1949	Fruggiero, Enzo, Cambridge	University of Rome
1949	Gardner, Carl Clinton, Jr., Framingham	Vanderbilt University
1949	Gardner, Frank Herbert, East Natick	Northwestern University
1949	Gary, John Everett, Cambridge	University of Oregon
1949	Gemmellaro, Salvatore P., Beverly	Long Island College of Medicine
1950	Gervais, Russell F., Natick	University of Vermont
1950	Gessay, Louis H., Braintree	Tufts
1950	Gigot, Albert F., Boston	McGill University
1950	Gilchrist, John J., South Boston	Tufts
1949	Giles, William P., Boston	Tufts
1950	Gleason, Elizabeth H., Wareham	Tufts
1949	Goldfarb, Abraham, Townsend	Midwest
1950	Golia, Ernest A., West Medford	Tufts
1949	Gottlieb, Stanley, Medway	Middlesex
1950	Graf, Helen J., Southbridge	Tufts
1950	Graham, James H., Melrose	Tufts
1949	Grant, Arthur Adolph, Lawrence	University of Vienna
1949	Gregorie, Joseph, East Boston	Middlesex
1949	Gross, Isadore M., Waltham	Middlesex
1949	Halperin, Meyer Herman, Boston	Long Island College of Medicine
1949	Hardin, Edmund B., Melrose	College of Medical Evangelists
1949	Harmon, Gaius E., Wilmington	Boston University
1949	Harvey, Norman Albert, Gloucester	Long Island College of Medicine
1949	Hayden, Thomas Henry, Pittsfield	University of Buffalo
1949	Healey, Edward Joseph, Mattapan	Tufts
1950	Hebert, Joseph V. R., Holyoke	McGill University
1950	Holmes, Jack K., Arlington	Tufts

1950	Holt, Earl K., Jr., Westboro	Harvard
1949	Horan, Raymond A., North Chelmsford	University of Maryland
1950	Horlick, Lloyd M., Topsfield	University of Vermont
1950	Howard, Charles S., Wakefield	Tufts
1950	Hunt, William W., Bridgewater	Boston University
1949	Hyde, Robert Wells, Boston	University of Vermont
1949	Jackson, James Higginson, Brookline	Harvard
1950	Jacobson, Franklin H., Avon	Middlesex
1949	Jaques, William Everett, Boston	McGill University
1950	Johnson, Joel M., Jr., Melrose	Tufts
1949	Judson, Walter Emery, Boston	Johns Hopkins
1949	Kaiser, Alfred Joseph, Hatfield	Harvard
1949	Kattany, Edward Charles, Natick	University of Indiana
1949	Kavanah, James Stephen, Brookline	Boston University
1950	Keeler, Robert W., West Roxbury	University of Pennsylvania
1950	Keighley, Thomas F. J., Natick	University of Vermont
1950	Kellett, Mirle A., Boston	McGill University
1949	Kelly, Harry J., Worcester	University of Vermont
1949	Kendrew, Leroy John, Westfield	Cornell University
1949	Kenney, James Francis, Boston	Boston University
1950	Kerr, Walter Stratton, Jr., Boston	University of Pennsylvania
1950	Kobrosky, Milton L., Springfield	Middlesex
1949	Kollar, Charles Stephen, Springfield	Georgetown
1949	Kolozetski, John William, Lynn	Middlesex
1950	Komando, Edward W., Rockland	Kansas City University
1949	Kootsey, Joseph S., Melrose	College of Medical Evangelists
1949	Krasner, Bernard, Quincy	Tufts
1949	Kreplick, Sumner, Lynn	Tufts
1950	Kuenster, Earl J., Worcester	University of California
1949	Kulka, Johannes P., Boston	Johns Hopkins University
1949	Kunan, Richard Ernest, Norwood	Hahnemann Medical College
1949	Kurland, George Stanley, Boston	Harvard
1949	Lambert, Robert Tyler, Lawrence	Boston University
1949	Landau, David, Boston	Tufts
1949	Landy, Edwin S. L., Medford	Middlesex
1950	LaRochelle, Frederick T., Springfield	Johns Hopkins University
1949	Latorella, John, East Boston	Middlesex
1950	Leavitt, Henry E., Stoneham	Middlesex
1949	Leff, Jerome, Springfield	Middlesex
1949	Lehmus, Harold J., North Wilmington	New York University
1950	Lippitt, William H., Boston	Johns Hopkins University
1949	Longino, Luther A., Boston	University of Arkansas
1950	Lovezzola, Michael M., East Boston	Tufts
1949	Lowenthal, Milton	Chester
1949	Magee, Lincoln Joseph, Winchendon	Kansas City University
1949	Magill, Herbert Kelvin, Brookline	Columbia University
1950	Magno, Henry A., West Medford	Tufts
1950	Maislen, Sidney, Springfield	University of Vermont
1949	Mangano, Salvatore N., Cambridge	Tufts
1949	Mansour, George Joseph, Lawrence	Georgetown
1949	Mann, James, Dorchester	Washington University
1949	Marnoy, Sherwood, Chelsea	Middlesex
1950	Marsh, Carl G., Worcester	Kansas City University
1950	Mason, Edward A., Boston	Washington University
1950	Matzilevich, Benjamin, Natick	Tufts
1950	McDermott, William V., Jr., Boston	Harvard
1949	McGoodwin, Maceo Dunbar, Springfield	Middlesex
1949	McKeough, Robert Paul, West Medford	Tufts
1950	McLaughlin, William F., Dorchester	McGill University
1950	Meyer, Fred M., East Gardner	University of Halle, Germany
1950	Mikal, Stanley, South Boston	Tufts
1949	Miller, Harry H., Boston	Harvard
1950	Miller, Joseph M., Framingham	Harvard
1950	Minkel, Herbert P., West Roxbury	Tufts
1950	Mitchell, George W., Jr., Boston	Johns Hopkins University
1949	Mixter, Charles G., Jr., Boston	Harvard
1950	Montgomery, Donald E. A., Westboro	Tufts
1950	Morganstein, Maurice, Fall River	Midwest Medical College
1950	Musante, Thomas P., Whitman	Middlesex
1950	Myers, Gordon S., Boston	Harvard
1949	Myerson, David J., Brookline	Tufts
1949	Nabseth, Donald Clark, Boston	Harvard
1949	Nau, Otto Schaefer, Jr., Medford	Boston University
1950	Nemiah, John C., Boston	Harvard
1949	O'Brien, Frederick W., Jr., Boston	Tufts
1950	O'Donnell, John A., Fall River	Georgetown
1950	O'Neil, John T., Malden	Albany Medical College
1949	Oppenheim, David Julius, Boston	Tufts
1949	Ossen, Paul Irving, Quincy	Boston University
1950	Ouellette, Phillipe W., Worcester	Loyola University
1949	Pace, Omar Thomas, Springfield	Iowa State University

1949	Palermo, Joseph John, Revere	Middlesex
1950	Parker, Jack S., Boston	Harvard
1949	Pash, James Joseph, Lawrence	Middlesex
1950	Phillips, Saul H., Roxbury	Middlesex
1950	Perlo, Vincent P., Boston	Tufts
1949	Platt, Warren D., Springfield	Columbia University
1950	Piszczek, Edmund S., Westfield	Middlesex
1950	Porter, Huntington, Waverly	Harvard
1949	Posin, Herbert Israel, Boston	Boston University
1950	Raker, John W., Boston	Harvard
1949	Ravven, Robert M., Boston	Harvard
1949	Raymond, Bradley Hickox, Wrentham	Tufts
1950	Reiman, Earl A., Worcester	Boston University
1949	Reiner, Leopold, Boston	University of Vienna
1950	Rexford, Eveoleen N., Cambridge	University of Buffalo
1949	Rice, Willard Gardner, Worcester	Tufts
1949	Robinson, John Christie, West Springfield	University of Vermont
1949	Rodkey, Grant V., Boston	Harvard
1950	Rosen, Sydney W., Fall River	Middlesex
1950	Rosner, Alfred, Willimansett	University of Vienna
1949	Rowe, Carter Redd, Boston	Harvard
1950	Roy, Louis E., Southbridge	Tufts
1950	Rubin, Harold S., Worcester	Tufts
1950	Rubin, Julius, Canton	Middlesex
1950	Ryder, Claire F., Boston	Tufts
1949	Sack, Theodore, Boston	Harvard
1950	Salzman, Ferdinand, Boston	Long Island College of Medicine
1950	Scannell, Robert J., Cambridge	Tufts
1950	Schiff, Arthur F., Cambridge	Middlesex
1950	Schwartz, William B., Jr., Boston	Duke University
1949	Scott, David Henry, Beverly	Harvard
1949	Scully, Robert Edward, Brookline	Harvard
1950	Selsby, Irving I., North Adams	Middlesex
1950	Sher, Jack E., West Roxbury	Middlesex
1950	Simkin, Stanley, Pittsfield	Albany Medical College
1950	Slaughter, Fred M., Salem	Harvard
1950	Slesinski, Frank A., Worcester	Southwestern Medical College
1949	Small, Wilfred Thomas, Boston	Tufts
1949	Smith, J. Roy, Worcester	Columbia University
1949	Soldini, Enrico Celeste, Fitchburg	Universtiy of Vermont
1950	Spiller, Arthur E., Boston	Tufts
1950	Starr, George R., Jr., Duxbury	Tufts
1949	Stearns, Norman Stanley, Brookline	Boston University
1950	Stein, Harold J., Watertown	Johns Hopkins
1949	Steinsieck, Robert Townsend, Newton Centre	Columbia University
1950	Stevens, Leyland E., Framingham	Cornell University
1949	Stone, Anthony Joseph, Athol	Yale
1950	Stone, William, Jr., Cambridge	Columbia University
1950	Stokes, Gordon D., Fall River	Northwestern
1950	Suchecki, Mary J., Worcester	Boston University
1949	Sullivan, Philip Burdett, Haverhill	McGill University
1950	Tabor, Edward O., Lowell	McGill University
1949	Thompson, Russell F., Randolph	Middlesex
1950	Tilley, Robert F., Brookline	Tufts
1950	Towne, Ruth A., Roxbury	Tufts
1949	Tracy, Robert J., Pittsfield	Harvard
1949	Truitt, Ralph Manolo, Melrose	College of Medical Evangelists
1950	Udis, Sanford W., Fall River	Boston University
1950	Van Gordon, Donald J., Worcester	University of Minnesota
1949	Vaughan, Warren T., Jr., Boston	Harvard
1949	Wacks, Joseph S., South Braintree	Middlesex
1949	Wagenbach, William F., Methuen	Kansas City University
1949	Wakefield, Horatio Paul, Norfolk	University of Vermont
1949	Wall, Roscoe L., Jr., Worcester	Jefferson Medical College
1950	Wallace, Edward P., Greenfield	Harvard
1949	Wallace, Joseph S., Boston	Boston University
1949	Warhaftig, Melvin Victor, Springfield	Middlesex
1950	Warren, Mary L., Bridgewater	Boston University
1950	Watson, Charles W., Boston	Yale
1950	Weed, Charles H., Framingham	Harvard
1950	Welch, Kenneth J., Waban	Harvard
1950	Wellington, Mark S., Framingham	Harvard
1949	Wheatley, Frank E., Jr., Beverly	Tufts
1949	Wheeler, Edwin O., Cambridge	University of California
1950	Wheelock, Frank C., Jr., Brookline	Harvard
1949	White, Samuel Eli, North Andover	Middlesex
1949	Whiting, Lewis Wood, Springfield	Western Reserve University
1949	Wickman, Robert Samuel, North Adams	Long Island College of Medicine
1950	Wildman, Chaim J., Pittsfield	University of Vienna
1949	Williams, Albert Harold, Revere	College of Physicians and Surgeons, Boston
1949	Williams, Lysle Westley, Amesbur	College of Medical Evangelists

1949	Willis, George Gibson, Framingham	Jefferson Medical College
1950	Winerip, Bernard B., Revere	Middlesex
1950	Wolfson, Irving M., Rutland	Yale
1949	Wood, William Leo, Lynn	Tufts
1949	Woodruff, Lorande Mitchell, Brookline	Yale
1949	Wright, Harold Stewart, Jr., Haverhill	New York University
1949	Wu, William Q., Cambridge	University of Michigan
1950	Zukel, William J., Newton Centre	Hahnemann Medical College

Deaths Reported from May 23, 1949, to May 16, 1950

ADMITTED	NAME	PLACE OF DEATH	DATE OF DEATH	AGE
1910	†Adriance, Vanderpoel	Williamstown	Some time in 1949	80
1926	Akin, Moses	Ellenville, New York	July 11, 1949	72
1920	Allen, Harold M.	Lawrence	July 3, 1949	59
1937	Benjamin, H. Weston	New Britain, Connecticut	May 4, 1949	47
1938	Bernhardt, Henry M.	Andover	August 31, 1949	41
1908, 1927	Boos, William Frederick	Fall River	August 11, 1949	79
1907	Burbeck, Edward K.	Marblehead	July 1, 1949	69
1910, 1915	Burns, Robert F.	Fitchburg	June 5, 1950	67
1908	Chase, Gilman L.	Clinton	March 16, 1950	70
1903	Clark, Thomas F.	Taunton	July 24, 1949	81
1933	†Coffin, Susan M.	Cochituate	June 1, 1949	69
1923	Condrick, James F.	Quincy	May 27, 1949	64
1906	†Cyr, Emile E.	Lawrence	June 13, 1949	72
1911	Dobson, William M.	Groton Long Pt., Connecticut	May 14, 1949	66
1929	Donovan, Arthur B.	Newton Centre	June 21, 1949	51
1913	†Dunn, William A.	Boston	July 14, 1949	?
1902	Dutton, Richard	Wakefield	March 10, 1950	75
1909	†Emery, William C.	Dorchester	August 20, 1949	75
1921	†Fawcett, Deborah	East Walpole	January 1, 1950	78
1898	Fitzgerald, Clara P.	Worcester	November 3, 1949	75
1947	Foster, George B., Jr.	Cambridge	January 1, 1950	66
1912	Gardner, Edwin D.	Marion	October 12, 1949	65
1946	Gaspar, Shari B.	Palmer	Some time in 1949	49
1909	Giddings, Harold G.	Newton Centre	May 28, 1949	69
1926	Goff, Almon P.	Muskogee, Oklahoma	November 8, 1949	76
1923	Gosian, Moses	Brockton	March 11, 1950	63
1901	†Grandison, Wilfed G.	Charlestown	November 6, 1949	73
1919	Greenberg, Boris E.	Boston	February 7, 1950	64
1904	†Greene, Ransom A.	Waverly	October 4, 1949	68
1914	Gwinnell, Alfred W.	Allston	May 3, 1949	71
1907	Hanson, William T.	Stoneham	April 20, 1950	70
1923	Harlow, John S.	Boston	February 7, 1950	53
1903	†Harriman, Perley	Lynn	June 16, 1949	82
1908, 1915	†Harris, Lorne W.	Punta Gorda, Florida	November 9, 1949	71
1919	Hatt, Rafe N.	Honolulu, Hawaii	June, 1949	60
1900	†Hawes, Alfred T.	Lynn	December 1, 1949	75
1906	Heffernan, David A.	Boston	April 18, 1949	68
1910	Hepburn, James J.	Norwood	October 26, 1949	66
1935	Hewitt, Wright P.	Belmont	February 2, 1950	44
1912	Howes, Frank M.	New Bedford	December 22, 1949	64
1926	Hull, Ira B.	Newmarket, New Hampshire	July 9, 1949	66
1911	Hunt, Harold O.	North Falmouth	February 21, 1940	68
1927	Kaszett, Peter W.	Lynn	November 23, 1949	46
1912	Kelley, Clarence M.	Waverly	Sometime in 1950	66
1923	Kelly, John J.	Marlboro	December 16, 1949	71
1938	King, Francis B.	Derry, New Hampshire	July 10, 1949	?
1931	King, Francis G.	North Quincy	February 8, 1950	50
1935	Krobalski, Joseph	Swampscott	September 3, 1949	56
1901	Lambert, John H.	Lowell	September 11, 1949	74
1932	†Lang, Walter E.	Westboro	September 11, 1949	67
1947	Larkin, John Joseph	?	August 26, 1949	35
1916	Little, George T.	Uxbridge	November 15, 1949	72
1901	†Mandell, Augustus H.	New Bedford	September 3, 1949	81
1924	Mason, Robert L.	Wellesley Hills	September 20, 1949	53
1910	Mathews, Robert F.	Worcester	July 17, 1949	68
1908	McCarthy, Eugene A.	Fall River	December 7, 1949	68
1922	McCormick, William A.	New Bedford	February 21, 1949	57
1898	McDermott, William V.	Salem	January 16, 1950	77
1898	McFee, William D.	Haverhill	September 6, 1949	75
1893, 1912	McKenzie, John R.	Watertown	March 16, 1949	82
1922	Meachen, John W.	Boston	February 24, 1949	57

1912	Minot, George R.	Boston	February 25, 1950	65
1919	Mulhern, Joseph P.	Worcester	February 1949	57
1904	Murphy, Edward M.	Lowell	December 1, 1949	73
1897	Newell, Franklin S.	Boston	March 3, 1949	78
1902	Nickerson, John P.	West Harwich	May 30, 1950	74
1903	Norris, Albert P.	Cambridge	March 29, 1950	76
1900	†Patterson, William F.	Medford	February 23, 1949	83
1933	Perkins, Franklin A.	Dorchester	June 5, 1949	66
1910	Phipps, Cadis	Boston	December 5, 1949	68
1899	Place, Ralph W.	Somerville	October 26, 1949	63
1906	†Plunkett, Harry B.	Lowell	February 10, 1950	?
1899	†Reagh, Arthur L.	W. Roxbury	June 15, 1949	78
1910	Reid, William D.	Newton	September 29, 1949	64
1914	Robinson, Henry A.	Hingham	August 21, 1949	62
1906	Rockwell, Alfred E. P.	Shrewsbury	June 19, 1949	79
1920	Rowland, William D.	Boston	July 1, 1949	70
1895	†Royal, Herbert B.	Harvard	September 14, 1949	86
1913	†Ruel, Joseph A.	Bradford	July 30, 1949	70
1906	Rushford, Edward A.	Salem	October 25, 1949	66
1898	Sabine, Jane D. K.	Boston	February 28, 1950	?
1924	Schwartz, Maurice R.	Fall River	February 20, 1950	68
1913	Sewall, Edgar F.	Somerville	March 22, 1949	64
1907	Shaw, Adam E.	Lowell	April 11, 1950	68
1938	Shibel, Edward.	North Adams	August 15, 1949	45
1921	Simpson, Charles M.	Temple, Texas	March 29, 1950	55
1915	Smith, Ernest E.	New York, New York	June 16, 1949	59
1931	Stochaj, John W.	Webster	October 28, 1949	55
1921	Story, Theodore L.	Southbridge	August 9, 1949	61
1901	†Sullivan, John J.	Dorchester	December 9, 1949	80
1939	Symington, Clifford L.	Westfield	November 1, 1949	45
1930	Tauro, Emily C.	Stoneham	June 23, 1949	48
1897	†Thomes, John B.	Pittsfield	December 5, 1949	79
1938	Thompson, Alfred P.	Wollaston	February 26, 1950	79
1921	Tierney, Thomas F.	Hudson	July 24, 1949	72
1912	Titus, Raymond S.	Boston	December 25, 1949	66
1901	Townsend, David	Bristol, Tennessee	February 1, 1950	76
1922	Vinal, Charles R.	Turners Falls	October 16, 1949	68
1897	†Washburn, Frederic A.	Boston	August 20, 1949	80
1907	Watters, William H.	Boston	October 10, 1949	73
1930	Weiss, Bernard	Mattapan	March 27, 1950	64
1924	Wilder, Ella A.	Middletown, Connecticut	July 20, 1949	52

†Retired fellow

Total number of deaths of active fellows	81
Total number of deaths of retired fellows	21
Grand total	102

The New England
Journal of Medicine

Formerly
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Established 1828

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The Journal does not hold itself responsible for statements made by any contributor.

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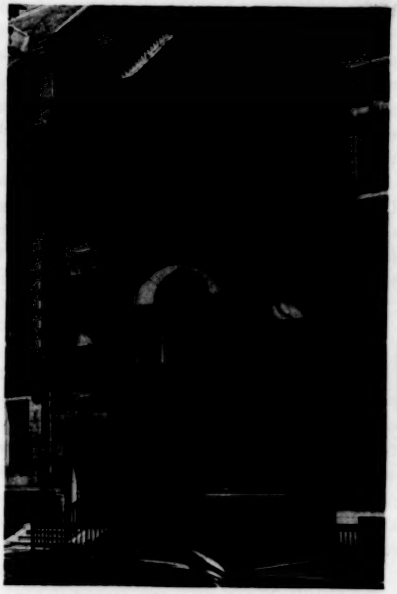
MASSACHUSETTS MEDICAL SOCIETY FINDS A HOME

After a hundred and sixty-nine years of corporate existence with no quarters or with rented ones, and certainly, in recent years, with inadequate ones, the Massachusetts Medical Society has finally acquired by purchase the building at 22 Fenway, adjacent to the Boston Medical Library. In this former mansion, after suitable alterations have been made, the Society will make its home, with adequate business offices, committee rooms, facilities for the Auxiliary, a hall for small meetings and ample space even for such functions as the Cotting Luncheon for councilors!

The Committee on Society Headquarters, which has been working actively on this particular and growing need of the Society, is to be congratulated on its final wise decision; it is further to be commended on the care with which, during the past year, it has explored the various possibilities that have been suggested for the solution of its problems. These possibilities have included such noble projects for the Society as joining the Boston Museum of Science and moving, in company with the Boston Medical Library, to the Charles River Dam; and of casting in its lot with other medical institutions



in the erection of a *Civitas Hippocratica* along the upper reaches of the Charles River or in some cloistered section of the Fenway more remote from the haunts of man than the present location of the Library and the offices of Society and *Journal*. In this search the technical assistance and advice of Mr. Herbert L. Beckwith, professor of architecture at Massachusetts Institute of Technology, has been invaluable.

Early in the deliberations of the Committee the decision was made that strength more than ever

resides in union; that the Boston Medical Library, the Society and the *Journal*, which constitutes the Society's largest single activity, should remain together for their mutual advantage and for the support of high standards of medical practice and education in this area.

Under the new arrangement the *Journal* will continue to occupy its present quarters in the south wing of the Library, a doorway being cut through from the *Journal* offices to the newly acquired building. The present physical association will then be made relatively permanent, to the advantage of all concerned, for the *Journal* draws heavily on the resources of the Library in its daily editorial work, and the Medical Library must still depend on the support of the medical profession for the very continuance, as well as the broadening, of its services.

An academy of medicine is coming into being in fact, if not in name; this new permanence of setting must spur the friends of the library on to providing for it the new stacks and other improvements that have been so badly needed for so long a time.

OCTOBER REVOLUTION

THE American Medical Association has announced plans for a climax in October to its National Education Campaign. The campaign, after nearly two years under the expert guidance of Whitaker and Baxter, will then move into its most critical phase, consisting of a nationwide advertising program in newspapers and magazines and over the radio.

This program, it is predicted, will cover 353 radio stations, 30 national magazines and all bona-fide daily and weekly newspapers, "irrespective of size, location or editorial policy." The schedules, which will run through the week of October 8, will be handled by four advertising agencies. The cost will be $1,110,000.

The campaign of the medical profession against compulsory so-called "health insurance," which has been apparently successful so far, has met with about the expected amount of criticism, both from its natural opponents who have other axes to grind and from the outspoken doubters and those of questioning minds within the ranks. These criticisms have been directed against the idea of a profession of service, intolerant of mercenary considerations, paying to carry its case to the people; they have been directed against the methods used, despite the general impression that the methods have been on the whole reasonably consistent with the dignity of even this austere profession.

In winding up the present show with a final and costly pre-election salvo, risks are being taken; but they are calculated risks and must be accepted. Such a burst of publicity will be highly criticized, but the criticisms will come from about the same elements that have been critical from the beginning; they will simply provide a new record to put on the noise machines.

The American Medical Association is presumably justified in embarking on a campaign of this nature and in accepting its cost. The important consideration is that the program must be above reproach in dignity and candor. No statement that is made can be susceptible to challenge, for in the quality of this program the reputation of the profession for maintaining certain ideals will be at stake. The Association must emerge from the campaign on better relations with its public than when it entered. Its record for straight dealing and for devotion to its principles must be a matter of public acceptance.

HORMONES IN ARTHRITIS

IN MAY, of this year, the price of ACTH and Cortisone was considerably lowered. The reduction in cost, however, reflects improved methods of producing these drugs, rather than their entrance into the pharmaceutical market. Although they are now available for more extensive testing, their use is still confined to clinical investigation and subject to restrictions imposed by Section 505 of the Federal Food and Drug Act.

Already, reports on ACTH and Cortisone have an obvious influence on medical meetings and literature. Many of these studies concern physiologic effects that, indeed, appear to be broad and profound. It is not surprising, therefore, that more than 50 clinical entities have been screened for their susceptibility to these drugs. In over 30

diseases or syndromes, some action has been noted, the fundamental and clinical aspects of this work having recently been summarized in the *Journal*.[1]

Most patients under experimental therapy with cortical steroids and related compounds belong to the group afflicted by diseases of mesenchyme. It is pertinent, therefore, to consider the clinical results in this category. May one expect that Cortisone and ACTH will be useful in the general treatment of rheumatic disorders and especially in that of rheumatoid arthritis? Has knowledge of that disease been increased by the application of these hormones?

So far, clinical investigation with ACTH and Cortisone has failed to show either the etiology or the pathogenesis of rheumatoid arthritis. The possibility of an infectious cause has not been eliminated. The postulate of an endocrine deficiency remains in doubt, and the theory of a hyperergic disturbance is still unproved. Studies of metabolic balance on patients with and without treatment have failed to elucidate the intermediary pathologic mechanism. The hormones have not cured rheumatoid arthritis, nor has administration of ACTH and Cortisone, by any mode known today, proved a practical means of therapy.

Such emphasis on the negative need not minimize the impressive changes that can be observed in rheumatoid patients receiving ACTH or Cortisone. The clinical manifestations of active arthritis diminish in a manner of time, degree and consistency not hitherto associated with any therapeutic plan or agent. Careful analysis suggests that this improvement in some but not in all cases equals that which has long been known to occur in so-called spontaneous remissions. Another parallel to the exacerbating course of rheumatoid arthritis may be seen in the flaring up of active disease after ACTH or Cortisone has been withheld for but a few days. This has been true in most cases, whereas continued defervescence of the rheumatoid state is rare after Cortisone as it is in the untreated patient.

The toxic effects of antirheumatic doses of these hormones represent, in part, extensions of their physiologic action; in part, they have not been explained. In any event they are sufficiently frequent and unpredictable to make a formidable deterrent to general clinical use. No reliable indicators of an incipient crisis associated with therapy have been established. Empirical adjustment of dosage and treatment schedules may conceivably result in a satisfactory method of controlling rheumatoid arthritis and of escaping harmful effects. The chances are greater that prolonged administration of these drugs, and several years of observation will be necessary before their practical significance in the field of rheumatic diseases can be assessed. By demonstrating the effect of Cortisone on rheumatoid arthritis, Hench and his associates[2] brilliantly advanced the method of studying this disease. Observations on the natural history of rheumatoid patients can now be amplified and checked against those on patients in an experimental situation.

Meanwhile, physicians will do well to rely on measures of proved value for the treatment of their rheumatic patients. Much harm and disappointment may be avoided if the temptation to premature use of experimental agents is resisted. This applies to Cortisone and ACTH in one sense, and in another to a number of steroids for which benefit to arthritis has been claimed with insufficient evidence. In the experience of investigators familiar with rheumatoid arthritis, Pregnenolone, 21-acetoxy pregnenolone and the gonadal steroids have not been promising. There is a clear indication for generous teamwork among investigators in several fundamental and clinical fields, all of whom are concerned with this important work.

References

1. Thorn, G. W., et al. Clinical usefulness of ACTH and cortisone. *New Eng. J. Med.* 242:783-793, 824-834 and 865-872, 1950.
2. Hench, P. S., et al. Effect of adrenal cortical hormone 17-hydroxy-11-dehydrocorticosterone (compound E) on acute phase of rheumatic fever: preliminary report. *Proc. Staff Meet., Mayo Clin.* 24:277-297, 1949.

HONORS TO DR. ALLEN

National recognition of the achievements of an outstanding member of the medical profession is always a source of satisfaction to his colleagues. When the accolade is bestowed by a foreign country, the gratification is all the greater. Such an honor was the selection of Dr. Arthur W. Allen, of Boston, former president of the Massachusetts Medical Society, to deliver the Hunterian Lecture in London on June 27 at the sesquicentennial celebration of

the Royal College of Surgeons of England; his subject was "Current Trends in the Surgical Management of Lesions of the Colon." Dr. Allen received a further mark of esteem on June 30, when the Royal College of Surgeons of Edinburgh made him an honorary fellow.

The *Journal* heartily congratulates the Royal College of Surgeons of England on the memorable occasion of its one hundred and fiftieth anniversary, and notes with pride the double honor conferred on a leader of the medical profession in Massachusetts and a surgeon of international reputation.

Dr. Macrae, of Howrah, in India, has administered oxygen gas to cholera patients, who immediately fell into a sleep from which they awoke much refreshed and completely restored to health, with the exception of weakness.

Boston M. & S. J., *July 24, 1850*

MASSACHUSETTS MEDICAL SOCIETY

DEATH

Cross — Louis K. Cross, M.D., of Winchendon, died on July 5. He was in his seventy-fourth year.

Dr. Cross received his degree from Boston University School of Medicine in 1899.

A son and a daughter survive.

BOSTON MEDICAL LIBRARY

THE CARSTEN COLLECTION

During his professional life and over a long period, Professor Paul Carsten, a well known Berlin ophthalmologist, occupied his leisure time in assembling the literary works of physicians and books relating to medicine and the physician in literature. During the rise of Naziism in Germany, he was forced to leave his country and went to England and succeeded in taking his books with him. When the London Blitz became imminent, it was decided to preserve the collection by disposing of it outside England and the Continent, and the Boston Medical Library acquired it in 1940.

The collection consists of about 850 volumes, all in fine condition, preserved in modern, serviceable bindings and classified and listed in thirty-five categories. The books are practically all in the German language and form a unique collection. Much of the material is ephemeral and difficult to obtain under present world conditions. A considerable number of the books were put on the prohibited list by the Nazi Government and were consequently confiscated or destroyed. There are over seventy volumes on the travels of physicians, a splendid addition to the large section of such material in the Library.

In 1930 Dr. Carsten wrote a monograph, based on his collection, entitled *Literarisches aus der Medizin, Medizinisches aus der Literatur* (Berlin, 1931). The text is divided into five main parts: medicine in verse; travels of German physicians in war and peace; a cursory random survey of the physician in literature; the physician in modern literature; and the nurse in modern literature. There is a comprehensive index of authors and titles mentioned in the text. The collection is housed temporarily in the Prince Room and is available for inspection and use.

CORRESPONDENCE

DANGER IN THE USE OF CERTAIN INSECTICIDES

To the Editor: It is important for Massachusetts physicians to be aware of the hazard involved in the use of certain organic insecticides recently introduced into all types of dairy, agricultural and fruit-growing industries. A few of the materials are listed here with their trade names: hexaethyl tetraphosphate (HETP); bladan (hexotine); tetraethyl pyophosphate (TEPP); and O,O-diethyl O-p-nitrophenyl thiophosphate (Parathion).

These materials are organic phosphates that are potent anticholinesterase agents. It is important to know that the mode of entry of the organic phosphates may be the unbroken skin as well as inhalation. The physiologic effects are those of stimulation of the parasympathetic nerves such as might be produced by an overdose of physostigmine. Since this is true, the specific treatment is the administration of atropine sulfate. The dosage depends on the severity of the symptoms and will range from 0.65 to as much as 2 or 3 mg. administered intramuscularly at hourly intervals if necessary. One large concern manufacturing organic phosphate insecticides is advocating that farmers keep twenty tablets of 0.65-mg. of atropine sulfate on hand for emergency use. This practice should be condemned because of the fact that atropine sulfate is poison and would certainly be administered mistakenly by laymen fearful of organic phosphate intoxication.

Morphine is contraindicated.

The reported deaths from organic phosphates have followed pulmonary edema, cardiac standstill or accident due to visual disturbance. The symptoms in order of frequency are anorexia, nausea and vertigo, insomnia, headache, abdominal cramps, diarrhea, sudden hot flashes, poor distant vision, increase in frequency of urination, drowsiness and mental confusion. The physical findings in order of prevalence are increased sweating, restlessness, tremor, increased salivation, dyspnea, pin-point pupils, bradycardia and evidence of pulmonary edema.

Because of deaths in the country as a whole, both in the manufacture of organic phosphates and in their use, warnings have been broadcast from the companies making these materials, from agricultural authorities and from federal

and state industrial hygiene agencies. However, it seems timely to present this specific material since case reports of toxic effects of organic phosphates used as insecticides continue to appear.

HARRIET L. HARDY, M.D.
Consulting Physician
Division of Occupational Hygiene
Massachusetts Department of Labor and Industries

BOOK REVIEWS

Selective Partial Ablation of the Frontal Cortex: A correlative study of its effects on human psychotic subjects. By The Columbia-Greystone Associates, Fred A. Mettler, M.D., Ph.D., editor. 8°, cloth, 517 pp., with 122 figures and 91 tables. New York: Paul B. Hoeber, Incorporated, 1949. $10.00. (*Problems of the Human Brain.*)

This informative, well written book offers the report of the first Columbia-Greystone Project designed to ascertain the therapeutic results of the operation for the mentally ill known as topectomy, and also to attempt to elucidate the mechanism by which any improvement occurs after psychosurgery.

Patients received a thorough medical, psychologic and psychiatric evaluation, both preoperatively and postoperatively. Postoperative changes were recorded in detail. The amount of study this group received was most comprehensive. The series is comparatively small, and the authors wisely do not attempt to draw any hard-and-fast conclusions. The data are presented, and the reader can draw his own general conclusions, which the authors believe may be as valid as any they might evolve. They are aware of the limitations of the data and are content with the belief that they have been able to dispel a certain number of misconceptions about the frontal cortex and that future work will be able to move forward more rapidly and certainly. In other words, the book describes their observations of patients with certain portions of the frontal cortex removed, in the hope that these observations will be used in future studies on the frontal lobe.

Some of the findings might be of more than passing interest. It was remarkable that so few histologic abnormalities were encountered in spite of the chronicity of many of the cases and the vigorous shock treatment that most of the patients had received. Whatever nerve-cell losses were observed appeared to bear no apparent relation to the type of case or duration of illness, treatment or area of brain examined. There was no evidence that the threshold for pain was increased; indeed, there was a suggestion that it was decreased. This is especially interesting in view of the fact that similar brain operations have been suggested for aid in control of pain from malignant growths.

In general, this book is worth while and should be read by all who are interested in brain function or surgery of the brain. It certainly should be in the libraries of all interested in psychosurgery.

Diseases of the Heart. By Charles K. Friedberg, M.D., 8°, cloth, 1081 pp., with 79 illustrations. Philadelphia: W. B. Saunders Company, 1949. $11.50.

This book is an important addition to the reference volumes concerned with cardiovascular disease. Its approach is essentially physiologic, and, indeed, Part 1, under the heading of "Circulatory Failure," constitutes an excellent physiologic textbook on circulatory dynamics. The other major divisions of the book, of which there are six, are cardiac arrhythmias, coronary circulation and disturbances in cardiac blood supply, structural abnormalities of the heart, etiologic forms of heart disease and special problems in heart disease. Extensive bibliographies are appended to the chapters. The seventy-nine illustrations are confined to electrocardiograms, phonocardiograms and roentgenograms. Electrocardiography, including description of the Vector cardiogram, is adequately discussed. Therapy is also emphasized in its modern aspects, including a mention of the preliminary report of the use of ACTH in the treatment of rheumatic fever. The title indicates correctly that the book does not cover the field of the peripheral circulation, nor does it have the same clinical emphasis of the older textbooks. There is no reference to auscultation or stethoscope as such in the index, and the eponymic references are confined to nine in contrast to sixty in the index of a similar book published only two years ago. The volume is recommended for the libraries of internists and cardiologists.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Human Growth: The story of how life begins and goes on. (Based on the educational film of the same title.) By Lester F. Beck, Ph.D., associate professor of psychology, University of Oregon. With the assistance of Margie Robinson, M.A. 8°, cloth, 124 pp., illustrated. New York: Harcourt, Brace and Company, 1949. $2.00.

This semipopular manual, written for teen-agers, parents, and teachers, presents the fundamentals of sex education for young people in simple, frank, understandable language. The text is well written, and the publishing is excellent. The title does not indicate the scope of the book. The volume should be in all collections on sex education.

Handbook of Medical Management. By Milton Chatton, M.D., instructor in medicine, University of California Medical School, Berkeley–San Francisco; Sheldon Margen, M.D., clinical instructor in medicine and research associate in medicine, University of California Medical School, Berkeley–San Francisco; and Henry D. Brainerd, M.D., assistant clinical professor of medicine and pediatrics, University of California Medical School, Berkeley–San Francisco, assistant clinical professor of pediatrics, Stanford University School of Medicine, and physician in charge, Isolation Division, San Francisco Hospital. 12°, cloth, 476 pp., illustrated. Palo Alto, California: University Medical Publishers, 1949. $3.00.

This handbook of medical treatment covers the whole field of medicine and is devised for ready reference. It is unfortunate that valuable material has been printed on the covers. When the small volume is used with the finding index this material will eventually be obliterated. Otherwise, the publishing is good.

Biological Antioxidants: Transactions of the third conference, October 7-8, 1948, New York, New York. Edited by Cosmo G. Mackenzie, Department of Biochemistry, Cornell University Medical College, and others. New York, New York: Josiah Macy, Jr., Foundation, 1949. $2.70.

This third volume of an important series contains ten short papers on antioxidation in its various aspects, including discussions on the chemistry and metabolism of vitamin E, the lipids, estrogens and androgens, flavonoids and tocopherol.

Metabolic Interrelations: Transactions of the first conference, New York City, February 7-8, 1949. Edited by Edward C. Reifenstein, Jr., M.D., Sloan-Kettering Institute, New York City. 8°, paper, 193 pp. New York City: Josiah Macy, Jr., Foundation, 1949. $2.95.

This new conference of the Josiah Macy, Jr., Foundation brought together twenty-five physicians interested in the problem of metabolism. The volume is well published and should be in all medical libraries as should all publications of the Foundation.

Blood Clotting and Allied Problems: Transactions of the second conference, January 24-25, 1949, New York, New York. Edited by Joseph E. Flynn, Department of Pathology, Columbia University, New York, New York. 8°, cloth, 231 pp., with 29 illustrations. New York: Josiah Macy, Jr., Foundation, 1949. $2.25.

Nineteen authorities in the special field of blood clotting participated in this conference. The publishing is excellent, and the series is recommended for all medical libraries and to all physicians interested in the subject.

Helpful Hints to the Diabetic. By William S. Collens, M.D., chief of the Diabetic Clinic, chief of the Clinic for Peripheral Vascular Diseases, and associate attending physician, Maimonides Hospital, attending physician, metabolic diseases, Jewish Sanitarium and Hospital for Chronic Diseases, Brooklyn, New York, and consultant in metabolic diseases, Rockaway Beach Hospital, New York City; and Louis C. Boas, M.D., chief of the Diabetic Clinic, associate visiting physician, Greenpoint Hospital, associate physician, metabolic diseases, Jewish Sanitarium and Hospital for Chronic Diseases, adjunct in medicine, Beth El Hospital, and assistant physician, Maimonides Hospital, Brooklyn. 8°, cloth, 135 pp., with 64 illustrations. Springfield, Illinois: Charles C Thomas, 1949. $3.00.

This small volume, written for diabetic persons and their families, supplies the information sufferers should have about the disease. A glossary of terms concludes the text, and there is a good index. The text is well printed with a large type. The book is recommended for medical collections in public libraries.

Cancer: New light on its causes, detection, treatments, cures and the brilliant promise of today's research. By Beka Doherty, A.B. 8°, cloth, 327 pp. New York: Random House, 1949. $3.00.

This book for the laity covers the whole field of the subject as it relates to the patient and physician and as a research and public-health problem. The text is well written and is based partly on information gained from well known authorities on cancer.

The appendixes are especially valuable since they bring together important information for physicians, librarians and the public. They include lists of approved cancer clinics, approved detection centers, and tissue diagnostic services in the United States and Canada. The book is well published and should be in all public and medical libraries.

Fractures and Dislocations in General Practice. By John Hosford, M.S. Lond., F.R.C.S., surgeon to St. Bartholomew's Hospital. Second edition revised by W. D. Coltart, M.B., B.S. Lond., F.R.C.S., assistant orthopaedic surgeon to St. Bartholomew's Hospital. 8°, cloth, 290 pp., with 87 illustrations. New York: Paul B. Hoeber, Incorporated, 1949. $5.00.

The first edition of this manual was published in 1939. The text has been revised with the objective of keeping the general practitioner informed of the up-to-date treatment of fractures and dislocations. A chapter on the fractures of the face and jaws by T. C. Henry has been added to this edition. An appendix presents brief historical sketches of five outstanding English surgeons who were eminent in the treatment of fractures, from Percivall Pott (1714-1789) to Edward H. Bennett (1837-1907). There is a good index, and the publishing is excellent. The book was printed in Great Britain.

NOTICES

HAMPSHIRE DISTRICT MEDICAL SOCIETY

The following is the program of the Hampshire District Medical Society for the next year:

Wednesday, September 6, 1950, at 4:30 p.m., at the Belchertown State School. Clinical Types of Oligophrenia. Medical staff of the Belchertown State School.

Wednesday, November 1, 1950, at 4:30 p.m., at Northampton State Hospital. Medical Malpractice. Dr. Walter W. Jetter, professor of legal medicine, Boston University School of Medicine.

Wednesday, January 3, 1951, at 4:30 p.m., at Veterans Administration Hospital, Northampton. Lobotomy. Medical staff of the Veterans Administration Hospital.

Wednesday, March 7, 1951, at 4:30 p.m., at Cooley-Dickinson Hospital, Northampton. Child Guidance. Dr. Olive A. Cooper, director of the Springfield Child Guidance Clinic.

Wednesday, May 2, 1951, at 4:30 p.m., at Jones Library, Amherst. Annual meeting and election of officers.

Dinner will be served after the meetings except on March 7.

INTERNATIONAL COLLEGE OF SURGEONS

The fifteenth annual meeting of the International College of Surgeons, United States Chapter, will be held in Cleveland, Ohio, from October 31 to November 5.

The program will include scientific sessions on subjects in the fields of general surgery; eye, ear, nose and throat surgery; gynecology and obstetrics; urology; and orthopedic, thoracic, plastic and neurologic surgery. In addition, an extensive technical and scientific exhibit will be presented by leading manufacturers of surgical instruments, x-ray apparatus, operating-room and hospital equipment, pharmaceuticals and others.

All doctors of medicine interested in surgery and its advancement are invited to attend, and can obtain a program upon request to Arnold S. Jackson, M.D., secretary, Jackson Clinic, Madison 4, Wisconsin. For hotel reservations, applications should be addressed to Committee on Hotels, International College of Surgeons, United States Chapter, 511 Terminal Building, Cleveland 13, Ohio.

AMERICAN SOCIETY FOR THE STUDY OF ARTERIOSCLEROSIS

The annual meeting of the American Society for the Study of Arteriosclerosis will be held at the Hotel Knickerbocker, Chicago, on November 5 and 6.

After registration from 8:30 to 9:30 a.m. on Sunday, November 5, the meeting will be devoted to sessions on various phases of experimental and clinical arteriosclerosis. The annual business meeting will be held from 2:00 to 2:30 p.m. on November 5.

SOCIETY MEETINGS AND CONFERENCES

JANUARY 9–DECEMBER 1. Laboratory Courses at Communicable Disease Center, Atlanta, Georgia. Page 950, issue of December 8.

AUGUST 21–26. International Society of Hematology. Page 888, issue of December 1.

AUGUST 28–SEPTEMBER 1. American Congress of Physical Medicine. Page 428, issue of March 16.

AUGUST 31–SEPTEMBER 7. World Federation for Mental Health. Page 306, issue of February 23.

SEPTEMBER 3–9. International Heart Congress. Page 160, issue of January 26.

SEPTEMBER 6–MAY 2. Hampshire District Medical Society. Notice above.

SEPTEMBER 7–9. All-Austrian Congress of Physicians. Page 994, issue of June 22.

SEPTEMBER 14. Progress in the Management of Hand Injuries. Dr. Henry E. Marble. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

SEPTEMBER 27. American Medical Writers' Association. Page xi, issue of July 13.

SEPTEMBER 27–29. Mississippi Valley Medical Society. Page 991, issue of December 15.

OCTOBER 12–14. American Association of Blood Banks. Page 428, issue of March 16.

OCTOBER 14–19. American Academy of Pediatrics. Page 962, issue o June 15.

OCTOBER 23–27. American College of Surgeons. Page xi, issue of June 29.

OCTOBER 23–28. Medicomilitary Symposium for Officers of Fourth Naval District. Page 34, issue of July 6.

OCTOBER 31–NOVEMBER 3. International College of Surgeons. Notice above.

NOVEMBER 1. New England Obstetrical and Gynecological Society. Hotel Somerset, Boston.

NOVEMBER 5 AND 6. American Society for the Study of Arteriosclerosis. Notice above.

NOVEMBER 6–8. New England Postgraduate Assembly. Hotel Statler, Boston.

JANUARY 31. American Academy of Orthopaedic Surgery (see Kappa Delta Award for Research in Orthopedic Surgery). Page 736, issue of May 4.

APRIL 9–13. American College of Physicians. Page 962, issue of June 15.

DISTRICT MEDICAL SOCIETY

HAMPSHIRE

SEPTEMBER 6–MAY 2. Program. Notice above.

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, AUGUST 3

THURSDAY, AUGUST 3

*9:00-10:00 a.m. Cardiac Grand Rounds. Main Amphitheater, Children's Medical Center.

FRIDAY, AUGUST 4

*9:00 a.m.-12:00 m. Combined Medical and Surgical Staff Rounds. Peter Bent Brigham Hospital.

(Notices concluded on page ix)